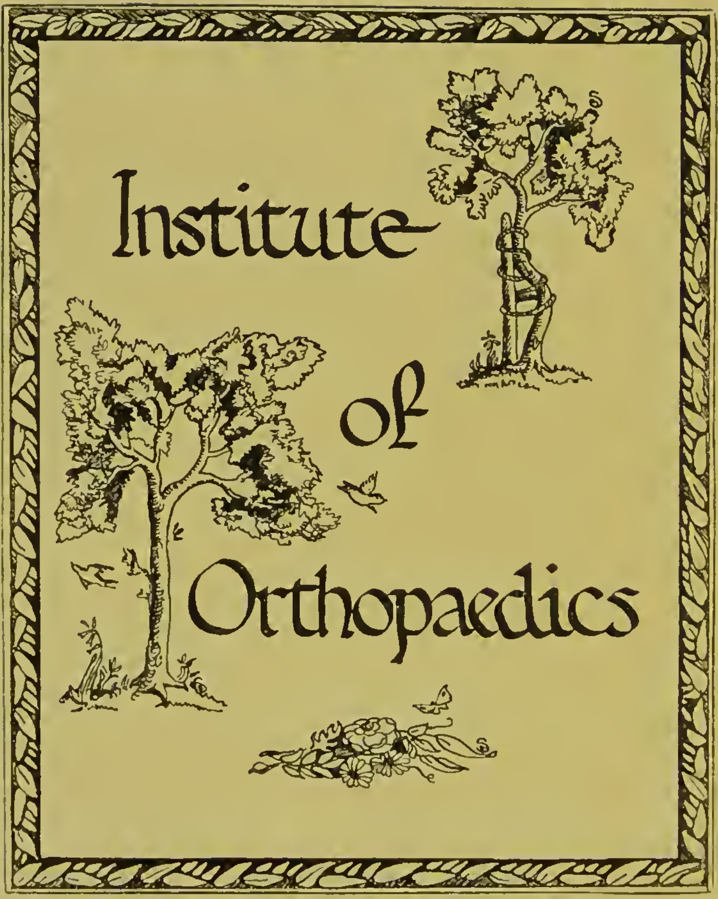
ARTHRITIS DEFORMANS

COMPRISING

RHEUMATOID ARTHRITIS, OSTEO-ARTHRITIS
AND SPONDYLITIS DEFORMANS

R. LLEWELLYN JONES



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RHEUMATOID ARTHRITIS, OSTEO-ARTHRITIS, AND SPONDYLITIS DEFORMANS.

[LLEWELLYN]

BY

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TO MY MOTHER'S MEMORY.

PREFACE.

The clinical material upon which this work is based is derived partly from experience gained during my medical officership to the Royal Mineral Water Hospital, Bath, and partly from that accruing from private practice at this Spa.

The chief aim of the book is to indicate and emphasize the essential distinctness of the two diseases—rheumatoid arthritis and osteo-arthritis.

Special consideration has been given to the premonitory symptoms which characterize the period of invasion of rheumatoid arthritis, in the hope that it may assist in that early recognition of the affection so essential for its successful treatment.

In the chapter devoted to the morbid anatomy of rheumatoid arthritis I wish to record my great indebtedness to Hoffa and Wollenberg's exhaustive *résumé*.

The classical descriptions of Virchow and Adams must inevitably be drawn upon by any writer on the subject of osteo-arthritis, but an attempt has also been made to set forth the more modern views as to its etiology, pathogeny, and treatment. Some stress has been laid upon the part played by "internal traumatisms" in accelerating the march of the disease in the affected articulations, while the indications for treatment arising out of this special feature, such as fixation of the joints, have been considered, likewise the more radical surgical measures recently advocated.

The vertebral type, or spondylitis deformans, has been described at some length in a separate section, with the object of supplementing the necessarily somewhat brief accounts met with in the ordinary text-books.

With this brief outline of the contents of this volume I now pass on to perform the pleasing duty of thanking those who have so generously assisted me in its production.

To my brother, Dr. Bassett Jones, of Aberystwyth, I am especially beholden, not only for unwearying assistance in collecting and reviewing the various articles scattered throughout English and foreign literature, but also for many valuable suggestions as to the arrangement of the subject matter—in short, for aid which can only be defined as indispensable.

Apart from acknowledging the great help I have derived from a study of Professor Pierre Marie and Dr. André Léri's well-known dissertations on the various types of vertebral ankylosis, I must also record my appreciation of the courteous and generous manner in which both these authorities placed their plates at my disposal.

In this connexion I must not omit to mention that *Plate XXXV*, as well as others, had previously appeared in the *Review of Psychiatry and Neurology*, to the editor of which journal, Dr. Alexander Bruce, I am indebted for the necessary blocks.

My grateful acknowledgments are also due to Professor Teissier, of Lyons, for permission to use *Plate XXXII*, which had previously appeared in the *Nouvelle Iconographie de la Salpêtrière* (1897).

To Dr. Higham Cooper, of University College Hospital, London, my cordial thanks are due for the excellent skiagraphs pertaining to osteo-arthritis, and to Dr. Bowker, of Bath, for those relating to rheumatoid arthritis and gout.

To the authorities of the Royal College of Surgeons, as well as those of University College Medical School, London, I am much indebted for the permission freely accorded me to study and photograph such of their pathological specimens as appeared relevant to the subject matter of this book.

My acknowledgments are also due to the Bath Corporation for kindly allowing me to obtain the plates relating to their thermal establishment.

My most grateful thanks are due to the Honorary Staff of the Royal Mineral Water Hospital, Bath, for the uniformly generous manner in which they granted me opportunities of studying the cases under their care.

In conclusion, to my friend Mr. W. M. Beaumont, of Bath, I tender my best thanks for much encouragement as well as for many valuable suggestions regarding this volume, which he has kindly seen through the press.

R. Ll. J.

40, Gay Street, Bath.
June, 1909.

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SECTION I.

GENERAL.

ARTHRITIS DEFORMANS.

SECTION I.—GENERAL.

CHAPTER I.

INTRODUCTORY AND HISTORICAL.

OF recent years it has become increasingly evident that before any solid advance in our knowledge of the chronic non-tubercular forms of arthritis can take place, a more exact nomenclature must be adopted. Various terms have from time to time been used to designate this mass of joint affections, which occupy, so to speak, an intermediate position between the frankly infective arthritides on the one hand, and the arthropathies of nervous origin on the other. Among the too numerous names now in use may be mentioned those of rheumatoid arthritis, osteo-arthritis, and rheumatic gout, as being most in vogue with English authors, while by the French physicians the terms rhumatisme noueux and rhumatisme chronique déformant are indifferently employed to denote these diseases.

By German authorities the term arthritis deformans is used to describe the condition of osteo-arthritis. In this book the term arthritis deformans will be used in a generic sense as covering all varieties of the affection, inasmuch as it has the advantage of being clinically descriptive of one or more morbid processes which, if not arrested, invariably deform, while this broad interpretation of its meaning does not conflict with any further subdivisions that may appear indicated. Latterly the opinion has been steadily gaining ground that not one, but several diseases have been included under one or other of the various terms used to designate collectively this class of joint diseases. The tardy appearance of any attempt at cleavage is very remarkable when we take into consideration the detailed accuracy with which the clinical characteristics of the two main sub-divisions, rheumatoid arthritis and osteo-arthritis, were described and differentiated by Charcot; indeed, as will be seen later, it is easy to discern in his and Vidal's writings a foreshadowing even of the pathological division recently advocated by Goldthwait into atrophic and hypertrophic arthritis.

One of the main obstacles to progress in the past undoubtedly arose out of our tacit adherence to this illustrious physician's conception of the essential pathological unity of all forms of the affection. As evidence of this do we not frequently see it inferred that osteo-arthritis is merely the later or more advanced stage of rheumatoid arthritis? What actual evidence have we that the atrophic or rheumatoid type does at any stage of its life history take on the characteristics of the hypertrophic or osteo-arthritic variety? Assuredly we have no pathological basis for this assumption; indeed, such as we have points to the fact that new bony formations, so specific a characteristic in osteo-arthritis, are invariably absent in rheumatoid arthritis, at all events in its early stages, and such as may develop later are inconsiderable, and not comparable either in size or character with those of osteo-arthritis, in which disease bony outgrowths appear early and are constantly present. It is true, as McCrae and others, including myself, have noted, that in any given case, while some of the joints may be of a rheumatoid type, other articulations in the same individual may be of an osteo-arthritic character, but such instances of the overlapping of the two varieties are comparatively rare, and as Goldthwait, who also admits this occasional co-existence, remarks, the articular lesions can usually be distinguished by their onset at different periods of the patient's life, as well as by their distinctive and widely differing characters. This existence side by side of rheumatoid and osteo-arthritic lesions cannot, however, be construed as a proof of their identity, or that one form may gradate into the other. We have an analogy afforded us by those instances in which the arthritic lesions of gout and osteo-arthritis are present in different joints of the same individual. We do not presume, therefore, that gout and osteo-arthritis are identical, or that one passes into the other, because of their occasional co-existence in the same patient. Clinically, as before alluded to, the essential distinctness of the two varieties of arthritis deformans has been tacitly acknowledged since the time of Charcot, and believing as I do, with Goldthwait and Garrod, that the two conditions are wholly diverse, they will be described under two headings—rheumatoid arthritis and osteo-arthritis. While it must be admitted that many are at present disinclined to accept this differentiation, I cannot help feeling that Goldthwait and Garrod's contention will ultimately prove correct. In order to settle this vexed question more speedily, it is highly desirable that pathological investigations should go hand in hand with careful clinical observations, as undoubtedly much of the delay in our progress is to be attributed to our having fallen into the error of dissociating the one from the other instead of checking and correcting, when necessary, our clinical inferences by our pathological findings, and vice versa. It is this lack of associated action on the part of the pathologist and clinician that has led us for so long erroneously to identify the clinical symptomatology of rheumatoid arthritis with the pathological lesions of osteo-arthritis. Recently, however, opportunities have been afforded us for examining, post mortem, the morbid changes that are to be met with in the early and active phases of rheumatoid arthritis, and these, together with the results of X-ray examinations, seem to indicate that our time-worn conception of the character of the anatomical lesions in this disease must be considerably modified, if not altogether discarded.

It is desirable therefore that full advantage be taken of any further chances that may offer for noting the early morbid changes that take place in the affection, in the hope that we may before long be in a position definitely to state what particular pathological lesions are distinctive of rheumatoid arthritis, as opposed to those we know to be characteristic of osteo-arthritis.

With this brief introduction we now pass on to consider our subject from its historical aspect.

As the "Sage of Norwich," in his treatise on Urn Burial, remarks, "Teeth, bones, and hair give the most lasting defiance to corruption," and were it not for this fact, that "Time, which antiquates antiquities, and hath an art to make dust of all things, hath yet spared these minor monuments," we, like sundry of our predecessors, might have been led to the erroneous conclusion that arthritis deformans was unknown among the ancients.

That no such immunity, however, was enjoyed by mankind, even in the most remote ages, has been conclusively shown by the researches of antiquarians who, aided by physicians, have detected unequivocal evidences of the disease in skeletons exhumed in the burial-places of bygone civilizations.

Strange to relate, not a few of these pathological relics are the fruit of excavations conducted in Egypt, a country to which, by reason of its dry climate, so many sufferers from this terrible malady at the present day resort.

Thus in 1890, Mr. Eve, at a meeting of the London Pathological Society, exhibited some ancient Egyptians' bones found in the tombs at Gurob by Mr. Flinders Petrie which presented the characteristic anatomical changes of osteo-arthritis. The tombs dated back to the Eighteenth Dynasty—about 1300 B.C.

More recently Dr. Page May described similar though more widespread lesions in another skeleton discovered by the same authority during his exploration of a cemetery at Deshasheh. In this instance, by aid of the pottery and scenic inscriptions upon two of the tombs, the date of the burial-place can be referred back still earlier, even to the Old Empire and to the Fifth Dynasty, namely, 3700 B.C. But it is to the labours of Professor Elliot Smith and Dr. Wood Jones that we are indebted for the most remarkable proofs not only of the existence, but also of the prevalence, of the affection amongst ancient peoples.

During the course of some excavations undertaken by the Survey Department of the Egyptian Government in that tract of Nubia lying immediately south of the First Cataract, over six thousand bodies were brought to light, comprising among them representatives of all periods from early predynastic times down to the fifth century after Christ.

As a result of their examination of this vast accumulation of human débris, Professor Elliot Smith, in the Nubian Survey Bulletin, states that "the disease which shows itself with by far the greatest frequency in the bodies of all periods is rheumatoid arthritis."

Again the ubiquity of the malady in olden times is shown by the fact that similar remains were discovered by Della Chiaje in the ruins of Pompeii, by Virchow in the precincts of a monastery at Marienthon, in Pomerania, by Dr. Norman Moore in a Roman sarcophagus situated in Smithfield, and lastly by Lebert in the Catacombs of Paris.

Now, while the mouldering bones exposed to view during these researches bear sure though silent witness to the antiquity of the affection, there is, unfortunately, nothing in the writings of ancient physicians, nor in those of a much later period, which would indicate that the condition was recognized clinically by them as a joint disorder distinct from others of the same category.

Indeed, looked at from an historical point of view, the clinical differentiation of the three diseases, gout, rheumatism, and rheumatoid arthritis, has been a most protracted process, extending itself literally over centuries.

The physicians of antiquity apparently enjoyed immunity from that zeal for infinite cleavage and subdivision so characteristic of the modern school.

Under the vague term, "articulorum passio," or arthritis, they grouped together these various affections, and under this title have handed down to posterity the clinical description of a disease in whose varied symptomatology we may sometimes trace the features of gout, sometimes those of rheumatism. Fortunately for us their clinical pictures were drawn with such vividness and accuracy that, despite this fusion of various joint diseases under one heading, students have been able to trace the salient features of at any rate one of them (gout) even in the writings of Hippocrates. Indeed, so far as gout is concerned we have little reason to complain of lack of literature, for as Charcot says, the productions of physicians, the works of historians, and the satires of poets, are full of allusions to this disease.

Cicero,[1] writing before the dawn of the Christian Era, describes the excruciating tortures of gout — "doloribus podagræ cruciari," and the peculiar burning character of its pains—"cum arderet podagræ doloribus." In the first century, Aretæus and Celsus described its characteristic features, while the Augustan poet,[2] in his Pontic epistles, laments that his gouty swellings defied the art of medicine. The works of Galen, in the second century, abound with information on the subject, and Lucian of Saramosta,[3] in his dialogues, enumerates the various remedies vaunted as specifics even in his day.

How widespread were the ravages of gout in the Roman world may be gathered from the fact that in the third century Diocletian, by an edict, exempted from the public burdens those severely crippled by it, and in the confusion of those days we may reasonably hope that some unhappy rheumatoid sufferers found a place amongst those who profited by this charitable dispensation.

From these remote times onwards through the Middle Ages to the present day an almost continuous series of historical records testify that not only has gout always been with us, but also that its clinical characters throughout the ages have remained unaltered, conforming always to the primitive type.

Charcot, in one of his lectures, reviews the antiquity of gout, and, while he pays a graceful tribute to the Greek and Roman physicians for their masterly disquisitions on this disease, deplores their silence on the subject of articular rheumatism, albeit the term itself, rheumatism, or rheumes as it was then spelt, dates from a very remote period. Used by the ancients more in accordance with its etymological sense, it finds a place even in the writings of Pliny[4] and Ovid,[5] but our modern conception of the disease differs widely from "the sharpe and eager flux of fleam which the Greekes call rheumes," or, more pertinently to our subject, "the flux of humours which the Greekes named rheumatism."

With the same significance it is used by the early English authors. Sir Thomas Elyot in his *Castel of Helth* alludes to the "reumes," and enjoins moderation in eating and drinking to those afflicted by them. It seems probable that the gradual extension of the term rheumatism so as to include muscular affections as well as catarrhal conditions was due to the fact, long recognized, that cold and damp are etiological factors common to both.

In *Julius Cæsar*[6] we read that Brutus was warned by Portia not to tempt the "rheumy and unpurged ayre" of night, while the following quotation from Dryden[7] exemplifies the broader use of the term rheumatism :—

> "The throttling quinsy 't is my star appoints,
> And rheumatisms ascend to rack my joints."
> *Palamon and Arcite.*

One cannot but admire the old poet's unwitting forecast of our modern views as to the relationship of tonsillitis to acute rheumatism.

The fact that the term rheumatism was for so long a synonym for fluxions, humours, and catarrhs of all sorts goes far to dissipate our surprise at the silence of the older physicians on the subject of articular rheumatism. Indeed, according to Charcot, their reticence on the point led several authors to assume that rheumatism was a modern disease, almost if not entirely unknown to the ancients. Such indeed was the opinion of the illustrious Sydenham, and later still this view was upheld by Hecker[8] and Leupoldt.[9] It seems, therefore, certain that the physicians of antiquity confounded articular rheumatism with gout, and this lack of discrimination on their part extended itself even to the acute forms of the two affections. Hallowed by tradition, this doctrine of their identity prevailed for centuries, in fact to the time of Baillon, who, according to Charcot, was the first physician who attempted to effect a cleavage. This observer,[10] dissociating the term rheumatism from its primitive interpretation, restricted its usage to that particular group of symptoms we now call acute articular rheumatism.

Considering the vast length of time that elapsed before acute rheumatism and acute gout were differentiated, it is not to be wondered at that the task, incomparably more difficult, of discriminating between the chronic forms of these two disorders has endured even to the present day. That our forefathers found the problem a difficult one may be gathered from one of their favourite sayings—"Rheumatismus agnatus podagræ." The phrase does not, as Trousseau points out, necessarily imply identity, though it postulates a near degree of relationship, and one may sympathize with, and at the same time admire, the astuteness of the old physician in such a quandary, in selecting a phrase which, while it indicates a difference, eludes a distinction.

In this old adage we may, I think, descry the beginnings of the more modern term—l'arthritisme—so beloved of the French authors, and in this connection we may mention that even as recently as the beginning of last century Trousseau's venerable predecessor at the Salpêtrière, Chomel, taught his pupils that gout and rheumatism were but different forms of the same disease; and still later do we find Pidoux[11] with no uncertain voice claiming that gout and rheumatism have but one common root, and so thorough-going was his advocacy that he brooked no exception, not conceding even acute articular rheumatism to be anything more than an expression of the arthritic diathesis. Charcot and Trousseau, although both convinced that gout and rheumatism were essentially different in nature, yet admitted that at the bedside the differentiation of the chronic forms was difficult if not impossible, and the former in one of his lectures points to the terms rhumatisme goutteux and rheumatic gout as tacit admissions of our powerlessness in this respect.

Hence it is easy to understand how the earliest clinical descriptions of rheumatoid arthritis appear sometimes under the title of chronic rheumatism, sometimes under that of chronic gout.

Thus in the writings of the illustrious Sydenham (1683)[12] we find an excellent account of its main clinical features under the term chronic rheumatism, of which he believed it to be an apyretic form. He pointed out that it differed essentially from gout, but that, like that disease, it might endure for a lifetime, its course diversified by remissions and exacerbations. In addition, he tells us that its excruciating pains, after lasting indefinitely, sometimes cease spontaneously, noting also that the joints are, so to speak, turned over, and that there are nodosities as in gout, especially on the inside of the fingers.

In 1703, under the title of "arthritis ex chlorosi," we find some undoubted examples of rheumatoid arthritis described by Musgrave,[13] a forecast of the view afterwards held by Forsbrooke, who considered anæmia etiologically related to the disease.

That John Hunter was fully acquainted with the anatomical character of the disease as it affects the knee joint, the following description, written by that great pathologist himself in 1759, testifies: "The articular cartilages have been removed from the greater part of the patella; the portions near the borders alone remain. The cartilage of the external condyle of the femur has been removed to a similar extent, and the surfaces of bone thus brought into contact and rubbed against each other in the movements of the joint are hardened, polished, and worn down in parallel grooves as if they had been chiselled. The remains of the articular cartilage on both bones are thin, and in many places degenerated into a thin fibrous substance; around their borders the bones are thickened and beset with projecting osseous plates and nodules."[14]

In 1764, Baron Haller,[15] in his *Elementa Physiologiæ*, describes an undoubted example of the disease as it affects the temporo-maxillary joint, noting the erosion of the cartilage and the presence of twenty small bodies (glebulæ) which were found contained within the capsule of this articulation.

According to Adams the first contribution to our knowledge of the pathological anatomy of the disease as it occurs in the hip joint we owe to the exertions of Edward Sandifort, of Leyden, who, in the second volume of the *Museum Anatomicum*,[16] published in 1793, delineated and described specimens showing the characteristic appearances. No name, however, was given to the disease by this author. This deficiency was supplied later by Sandifort junior,[17] who contributed specimens of the same disorder to the fourth volume of the same publication, stating that he considered the morbid changes due to rheumatic disease.

It was not until 1800 that Landré Beauvais[18] issued his thesis treating of the disease clinically under the name of "*Goutte Asthénique Primitif*," and most authorities concur in awarding the palm to this observer for being the first to differentiate the affection. Beauvais, however, as Dr. A. E. Garrod says, undoubtedly included cases of true gout, not to mention other obscure forms of arthritis, under the term. Still the words "Doit on admettre une nouvelle espèce de goutte?" go far to justify Charcot in his claim that Beauvais, despite the title of his thesis, fully recognized that the disease differed from gout.

A few years later (1804–16) Heberden,[19] in his Commentaries, wrote: "The disease called chronical rheumatism, which often passes under the general name of rheumatism and is sometimes supposed to be gout, is in reality a very different distemper from the genuine gout and from the acute rheumatism, and ought to be carefully distinguished from both." He gives a clear description of its clinical features, observing that "there was little or no fever, no redness of the skin, no great pain, but swelling of the affected part; that the disease was not particularly liable to begin in the feet. He also noted that one attack was followed by more weakness of the limbs than would have been produced by gout in many years, that the general health was affected, and that cramps were common." Heberden drew attention also to the extreme crippling induced by the disease and its protracted course, and his name to the present day is associated with the nodes on the terminal phalanges.

In 1805 the distinguished old Bath physician, Haygarth,[20] published his classical essay entitled: *A Clinical History of the Nodosity of the Joints*, the opening sentence of which shows us that, like many of his successors, he deplored the loose manner in which the term rheumatism was used and applied "to a great variety of disorders which, beside pain, have but few symptoms that connect them together," The name "rheumatick gout" he disapproved of as tending to perpetuate its confusion with gout and rheumatism, and suggested the term "nodosities," in the hope that "as a distinct genus it will become a more direct object of medical attention." Commenting on his series of thirty-four cases he says: "I find two where the knees only were attacked. In all, or nearly all, the rest, the hands, chiefly the fingers, were affected. These diseased joints generally suffered pain, especially at night, but in a less degree than might be expected from such a considerable morbid change. They feel sore to the touch. In one case the patient was attacked with severe spasmodic pains." Amongst other points noted by Haygarth was the especial liability of the disease to attack women, especially at the menopause, its crippling character, and the distortions and dislocations of joints produced in inveterate cases. He did not consider that the muscles were affected in the disease—a statement somewhat difficult to reconcile with our modern view that muscular atrophy is one of the most striking features of the affection. He gives us no positive views of his own on the pathology of the disease, but judging from the antiphlogistic treatment advocated by him we may assume that he deemed it of an inflammatory nature. The symptoms of the disease, to his mind, clearly differentiated it from acute and chronic rheumatism, also from gout, but, having observed that the nodosities in two or three of his cases appeared to alternate with a " colic of the stomach," and in one instance with " an asthma," he expressed his belief that if such examples " of the translation of the disease to and from the viscera be confirmed, they would prove that the nodes were in their nature more allied to gout than rheumatism."

He found a further resemblance to gout in the fact that the disease, in his experience, afflicted more commonly the higher and middle classes, and he concludes his reflections with a pious hope that they (the nodes) would not resemble gout in the difficulty of discovering efficacious remedies to cure or relieve it.

Subsequently to Haygarth's work no further contributions to our knowledge of the disease were made until the time of Cruveilhier. Chomel,[21] it is true, in 1813, in one of his works, does allude to the malady described by Beauvais, but only to remark that the affection was rheumatic in nature. Similarly we find Scudamore,[22] in 1827, stating that Haygarth's nodosities were but rarely seen save as an effect of gout or rheumatism.

Between the years 1829 and 1840, however, Cruveilhier,[23] in the *Anatomie Pathologique*, complains that while the disease, looked at from an anatomical point of view, had received ample attention, on the other hand, from the clinical side, it had been sadly neglected, and that he considered that the time had now arrived (to adopt his own words) for it " prendre rang parmi les maladies des articulations." This distinguished physician designated it " usure des cartilages articulaires," thus giving to the disease, as Adams says, a local habitation and a name. It must not, however, be assumed that Cruveilhier confined the destructive processes to the articular cartilages, for elsewhere he describes the affection as " une inflammation chronique de la synoviale."

Amongst other points this authority proved from dissections that bony ankylosis rarely occurred, a question on which, at the time, there existed considerable differences of opinion.

During this period, both on the Continent and in England, the morbid appearances created by this disease in the joints were being very frequently confounded with those due to trauma. Thus we find Adams, in 1831, in his clinical lectures at the Jervis Street Hospital, Dublin, drawing attention to such fallacies in regard to the disease when localized in the hip. and to which form he applied the term "malum coxæ senilis." Similarly, Robert Smith, in 1835, commented on the frequency with which some errors in diagnosis occurred, and this not only as regards the hip, but in reference to the shoulder joint also, and in an able memoir[24] he critically analyzed some twenty-four cases, brought forward by different authors as examples of injury to the shoulder, conclusively proving them all to have been examples of what he and Adams termed chronic rheumatic arthritis affecting this articulation.

Edwin Canton[25] did a like service in exposing identical mistakes in regard to the joints of the great toe. Even as late as 1850, he says, cases were brought before the Fellows of the Medical Society, London, and published in the *Lancet*, as examples of "luxation of the distal extremity of the great toe," notwithstanding that the morbid changes in the joints were perfectly characteristic of those of chronic rheumatic arthritis.

In France at this time the malady was being studied by Lobstein[26] (1833), Deville[27] (1848), and Broca[28] (1850), and in Germany by Meyer[29] (1849), Weber[30] (1858), and Leis.[31] Lobstein noted the fragility of the bones, the eburnation following destruction of the cartilages, and the presence of bony outgrowths besetting the edges of the articular surfaces. Deville and Broca worked at the morbid anatomy, completing in some respects the contemporary work of Adams, and to these authors we owe the origin of the term "arthrite sèche." The tissue modifications that ensue as a result of chronic rheumatism were elucidated by the work of Leis, Meyer, and Weber, and they were confirmed from the French school by Cornil,[32] Vergely,[33] and Ranvier[34],

The most prominent English workers were Colles (1839–1857), Brodie (1833), Redfern (1842), Todd (1843), Graves (1843), Fuller (1852), and Adams (1857). Colles,[35] in making a communication to the Dublin Pathological Society, called in question the inflammatory nature of the disease, saying, "If it be inflammation it has not the characters or consequences of true inflammatory action. This observer also showed "that two very opposite processes are to be found going on at the same time, viz., "absorption of the old bone and its cartilage of incrustation, with deposition of new bony matter." Brodie[36] saw in the disease resemblances to both gout and rheumatism, and termed it rheumatic gout, but nevertheless maintained that it was essentially distinct from both. In his experience it was more prevalent among the upper classes of society. He agreed with Cruveilhier in his view that the synovial membrane was the site of the earliest changes, and, like him, deemed them of an inflammatory nature, but differed from him in that he thought bony ankylosis frequently resulted in the affected joints.

In Todd's[37] reflections on the pathology of the affection we see the dawn of a reaction against the view assigning an inflammatory origin to the joint changes. Thus he writes: "The rheumatic affection of the joints may be most correctly described as an abnormal nutrition, occasioned by the presence of a peculiar matter in the nutrient fluid, affording doubtless certain points of resemblance to chronic inflammation, yet differing from it in a very marked degree." In this remarkable passage can be seen a very definite forecast of the trophic theory, all views of which are based upon some abnormality in nutrition of the affected joints.

Approaching the disease more from its clinical side, Graves[38] emphasized the fact that "cases of rheumatic arthritis, attended with prolonged excitement of the circulation, and copious sweating, are generally found to exhibit an intractable chronicity, and too often terminate in rendering the unfortunate patient a cripple for life." This profuse sweating, he goes on to say, never tends either to diminish fever or relieve pain, and he gives a striking example of its localization to a limited portion of the body surface in one of his patients. Indeed, so struck was he with its prominence as a clinical symptom that he gave to the affection the name of "the sweating arthritis."

To Redfern[39] we are indebted for an able contribution on the destructive changes that take place in the intra-articular structures, more particularly the cartilages. Fuller,[40] in his valuable monograph, refers to the disease under the title of "rheumatic gout," at the same time pointing out that "the natural history of rheumatic gout accords but little with that of true rheumatism, and is equally inconsistent with that of true gout." He considered that its frequent exacerbations, their increasing severity and obstinacy, coupled with the marked predilection of the disease for the smaller joints, sufficiently distinguished it from rheumatism, not to mention that the disease never involved the heart or its membranes. On the other hand, it contrasted with gout in that its victims were usually the young, the middle-aged, the slender, and the weakly, and moreover, it was much more common in women, and, unlike gout, it simultaneously invaded many joints.

In 1857, Adams's magnificent work appeared under the title of *Chronic Rheumatic Arthritis*. It would be difficult to overestimate the great value of his contribution to our knowledge of the morbid anatomy of the disease. Indeed, as Charcot says, from the point of naked-eye examination his description has left very little to be desired. He steadily defended Cruveilhier in his contention that the disease began in the synovial tissues, and that it was inflammatory in nature. Classifying his cases clinically into two main groups, the constitutional and the localized forms, he pointed out that the former, in his experience, commonly developed directly out of acute articular rheumatism, and occasionally resulted from puerperal arthritis, while the latter might arise as a result of injury. To this authority also are we indebted for the first forecast of the pathological division into the *atrophic* and *hypertrophic* types of rheumatoid arthritis on which such stress has recently been laid by many students of the disease. Nor did Adams neglect to note the severity with which the muscles are assailed, pointing out the severity of the spasmodic contractions and the intense atrophy, not only of the muscles themselves, but also of their tendons, which he showed in some cases underwent even total absorption.

He adduces several examples where such absorption of the intra-articular portion of the tendon of the biceps led to partial luxation of the shoulder joint and consequent confusion with the results of trauma. In one respect his observations have not been confirmed, namely, that the disease is equally common in both sexes, and in this particular he contrasts his experience with that of Haygarth, who, though he had been studying the disease for twenty-six years, only met with one instance of the affection in the male sex.

In 1853 appeared the important theses of MM. Charcot[41] and Trastour.[42] Both of these eminent physicians believed it to be a modification of rheumatism, maintaining that although as a rule "chronic articular rheumatism," to use their own nomenclature, developed insidiously, yet at the bedside they had seen this chronic form arise directly out of acute articular rheumatism, their testimony as to this transition of the one disease into the other according with that of Adams, Fuller, and Graves. The far-reaching scope of their united researches precludes even an attempt at a résumé of their work, which will be referred to seriatim under most of the sections throughout this book.

In 1855 Vidal's thesis[43] on the subject appeared, enriching materially our clinical knowledge of the malady. It is to this author that we owe the first clinical description of the atrophic type of the disease, and Charcot, in his clinical lectures, refers to it as the *atrophic form of Vidal*, noting that in this variety "induration of the skin, a sort of scleroderma, develops, the cutaneous covering is cold, pale, smooth, polished, and will not wrinkle"; adding also that in such cases atrophy of the bones and muscles accompanies the wasting of the soft tissues. This recognition clinically of the atrophic type by Vidal is extremely interesting when taken together with Adams's[44] more recent pathological observation that not only hypertrophic but atrophic changes might be met with in the bones when affected by this disease.

The view held by Charcot and Trastour that the disease was only a form of rheumatism has always held its own in France. It was supported also by Besnier,[45] Homolle,[46] Lacaze Dori,[47] and Mathieu;[48] and Charcot, even in his later writings, maintained his original opinion on this point. English authors, on the other hand, have, as a rule, remained faithful to Haygarth's and Heberden's conception, namely, that it was a disease quite distinct from gout and rheumatism.

This view was strongly supported by Fuller, Adams, and Sir Alfred Garrod,[49] the last-named showing conclusively that the excess of uric acid in the blood, so typical of gout, is absent in rheumatoid arthritis, and also maintaining with Fuller that the cardiac lesions of true rheumatism are not to be met with in rheumatoid arthritis. Sir A. Garrod, in the first editions of his work, retained the old term "rheumatic gout," and Fuller, in his writings, records with evident satisfaction that "even Dr. Garrod has deserted his favourite bantling rheumatoid arthritis and has adopted the term rheumatic gout." In his third edition, however, Sir A. Garrod definitely applied the term rheumatoid arthritis to the malady with which his name is so honourably associated. Classifying the disease under the three headings, acute, chronic, and irregular, he further subdivided the chronic type into two varieties, the generalized and the localized forms. The prevailing humoral theories as to the pathology of the disease were now profoundly altered, and this owing to important contributions to our clinical knowledge of the disease made by Remak, Senator, Sir Jonathan Hutchinson,[50] Sir Dyce Duckworth, Ord, Spender, and others. Hitherto, if we except the pathological views advanced by Todd, and held in a less definite manner by Colles, all authorities, excepting Remak, concurred in ascribing the joint changes to inflammatory or sub-inflammatory action. Remak,[51] however, in 1858, put forward the idea that the articular changes were secondary to, and dependent upon, some disorder of the nervous system. To some extent this suggestion of a neural origin received colour later on from Charcot's[52] statement that he had seen several cases of locomotor ataxy accompanying dry arthritis and nodosities of joints (rhumatisme noueux d'origine nerveuse). That Trousseau[53] had leanings towards some implication of the nervous system may be gathered from the following passage: "Are not the pains and muscular retractions observed in nodular rheumatism the result of the morbid cause rheumatism acting pathogenically upon the muscles and nerves at the same time as upon the joints?"

In 1876 this conception of its pathogeny was revived by Senator,[54] who, describing the condition under the term suggested by Virchow, i.e., arthritis deformans, considered that its symmetrical mode of onset and progress were most easily accounted for by some disturbance of the central nervous system, and this view was ably advocated later on by Sir Dyce Duckworth, Ord, and Spender.

While believing its victims to be free from any gouty, rheumatic, or scrofulous taint, Ord[55] contended that no specific origin could be claimed for the malady: in fact, that it was not to be regarded as an independent disease, but rather as a lesion common to many ailments. Dissenting from the humoral theory, he propounded a neural one, claiming (1) That the disease might be met with as a result of a primary lesion in the cord; (2) That it might arise reflexly from such varied sources of irritation as gonorrhœa, simple urethritis, utero-ovarian disorders, etc.

Spender,[56] out of his vast experience, earnestly supported the neural theory, claiming that the arthritis was only one sign of it among many others, and possibly not the most important. Regarding the arthritis as the central or substantial element of the disease, he would not allow that the collateral nervous phenomena were one whit less important. Consequently we find in his clinical pictures no lack of perspective. No one symptom is unduly emphasized. All alike—the muscular atrophies, the pigmentary changes, the vasomotor and trophic disturbances, together with the joint lesions—are treated impartially. In short, Spender's graphic description is not so much one of rheumatoid arthritis as one of a rheumatoid patient.

In 1890, Dr. A. E. Garrod, calling the condition rheumatoid arthritis, wrote a treatise on the subject in which he very ably advocated the dystrophic theory of the disease, maintaining that "it affords the best explanation of the peculiar features of rheumatoid arthritis, of the variety of its clinical forms, in spite of the identity of the articular lesions and of the characters and distribution of those lesions." He differed from Spender, however, in that he thought that the associated nervous phenomena, excluding certain early and premonitory symptoms, were secondary rather than primary phenomena of the disease.

In strong contrast to the views just cited are those advanced by Hutchinson, Arbuthnot Lane, and Pye Smith. Hutchinson[57] looked upon rheumatoid arthritis as a blend of the rheumatic and gouty diathesis in varying proportions, at one time the gouty element predominating, at another the rheumatic. Arbuthnot Lane,[58] on the other hand, refuses to regard the condition as a disease at all, contending that the local lesions are adequately accounted for by wear and tear; in other words, that they are simply the results of the pressure exerted by opposing articular surfaces upon each other, while Pye Smith[59] holds that at any rate in some cases of rheumatoid arthritis the articular lesions are the result of senile changes.

In 1890 appeared the work of Lane and Griffiths,[60] dealing particularly with the differential diagnosis of the three diseases, chronic rheumatic arthritis, rheumatoid arthritis, and osteo-arthritis.

Forsbrooke's[61] dissertation was published not long afterwards, the author assigning a condition of anæmia as the underlying cause of the vasomotor and trophic phenomena, and attributing the articular lesions to trophic disturbances of nutrition.

The year 1892 marked the commencement of a new era in our pathological study of the disease. Up to now the humoral and neural or dystrophic theories had held undisputed sway, but in this year Max Schüller[62] launched the microbial theory of its origin, basing his claim on the fact that he had discovered micro-organisms in the synovial fluid of the diseased joints. No further advocates of the infective theory appeared until the year 1896, when similar claims were brought forward almost simultaneously by Bannatyne and Wohlmann,[63] working in collaboration with Blaxall, and by Chauffard and Ramond.[64] In the following year the microbial theory was supported by Professor Bäumler,[65] who opened a discussion on the subject at the Berlin Congress for Internal Medicine. Amongst other conclusions arrived at were the following: "That it is not unlikely, and the more recent bacteriological researches give this idea a certain basis, that we are dealing with an infective disease, and that it should not be lost sight of that there may be several causes and infections at work at one and the same time." On the other hand, Chvostek, at the same discussion, was resolutely opposed to the bacterial theory, seeing no ground whatever for considering it to be of infective origin. Stewart,[66] at the meeting of the British Medical Association at Montreal, opening a discussion on the disease, strongly advocated the infective theory; Jacobi, of New York, joining in the same debate, suggested that the presence of cocci in a few cases did not prove them to be the cause; and Shattock, of Boston, drew attention to the fact that Dr. Goldthwait had been unable to confirm the observations of others as to the presence of micro-organisms in the synovial fluids. In the same year Riesman,[67] of Philadelphia, discussing the various views as to the nature of rheumatoid arthritis, inclined to the opinion that it was of nervous origin, as being most in consonance with the symptomatology.

Shortly afterwards Triboulet and Thomas[68] reported from Dejerine's clinic the case of a female, aged 19, who had suffered for nearly two years from chronic polyarthritis which dated from a puerperal infection, the autopsy of which revealed extensive change in the spinal cord affecting chiefly the posterior columns. In 1898 Von Dungern and Schneider[69] reported a case of chronic arthritis deformans from Bäumler's clinic in which diplococci were cultivated from the joint fluids. In the following year Collinson and Southam[70] reported cases in which excision of the affected joints had been attended by good results, and in 1900, Fernet and Lacapere[71] discovered pneumococci in the joint fluid of a woman suffering from pneumonia who afterwards developed rheumatoid arthritis.

In 1900, A. O. J. Kelly,[72] considering the pathogenesis of arthritis deformans, inclined to believe it to be of the nature of an infectious trophoneurosis. In 1901, Painter,[73] of Boston, in an able paper, critically analyzes the different views held as to the nature of the pathological changes met with in rheumatoid joints. Sharply dividing off rheumatoid arthritis from osteo-arthritis, he maintains that the histological changes observed in the articular lesions of the former could be accounted for on a neurotrophic basis, a view of their origin distinctly suggested by the clinical course and features of the malady. In the same year Preble and Hektoen[74] contributed a remarkable example of arthritis deformans in a young girl in whom the condition had supervened in sequence to multiple fibromata situated upon the various nerve trunks. Stengel,[75] reviewing this case, considered that it lent support to the nervous theory of the origin of arthritis deformans. In the following year Macalister,[76] of Liverpool, dealt with the mode of onset of acute rheumatoid arthritis, stating that certain protracted cases of rheumatic fever refractory to salicylate treatment passed on into a typical rheumatoid condition, while Poynton and Paine, in the same year, read a paper before the Pathological Society dealing with the experimental production of the osteo-arthritic type of arthritis deformans.

In reviewing this attempt to outline the differentiation of arthritis deformans from gout on the one hand, and rheumatism on the other, we note how tardily the work of cleavage has been achieved. On the pathological side we have seen how the time-worn humoral view gave place to the neural theory of its origin, while this again for the time being was almost displaced by the infective theory of its causation. At the present time there would appear to be an attempt to reconcile the long-standing controversy that has existed between those who uphold the microbic theory and those who favour the central origin of the disease. Teissier, for example, in his recent work, has put forward the view that some cases at any rate might be explained on the basis of an infectious or toxic trophoneurosis.

At present an early solution of the pathological riddle presented seems hardly to be expected, the bacteriological findings by different observers being so flatly contradictory, and the lesions in the nervous system so scanty and inconstant, that no view can be held to form a wholly satisfactory, much less a conclusive, explanation of the varied phenomena of the disease.

Again, on the clinical side we have observed how arthritis deformans slowly but surely asserted itself as an affection distinct both from gout and rheumatism, and in the course of our sketch we have also traced the growth of the modern opinion that at least two separate conditions, rheumatoid arthritis and osteo-arthritis, are comprised within that term.

Should future research conclusively establish the truth of this conception it cannot be doubted that it would constitute a great step forward in this slow process of differentiation of the various joint diseases grouped under that still vague and ill-defined term, arthritis deformans, which has in truth for so long been the dumping-ground for many alien joint disorders.

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Chapter II

CLASSIFICATION OF ARTHRITIS DEFORMANS.

It will be readily admitted that much remains to be done before a satisfactory classification of the various diseases grouped together under the term arthritis deformans can be formulated.

Doubtless, as all who have studied the extensive literature on the subject will agree, one of the chief obstacles to progress in this respect has consisted in, and still continues to be attributable to, the very varied nomenclature adopted by individual writers in describing the same types of disease. Needless to say, had the various terms used always been accompanied by a clear-cut definition or account of their clinical equivalents, much of the existing confusion would have been obviated. But such criticism must be tempered by the fact that the inherent complexity of the subject renders it only too easy for any student of the disease to have a quite clear-cut clinical impression himself of what he terms arthritis deformans, rheumatoid arthritis, etc., and yet be quite unable to convey it in unambiguous fashion to another mind.

It is only necessary to be present at any discussion on the subject to become painfully aware of this as a very real bar to progress, and one cannot help feeling that a frequent demonstration of cases at such assemblies would do more than any amount of discussion to dissipate the mutual misunderstanding which so hampers advance.

In the historical section I have alluded to the fact that it is to Adams[1] we are indebted for the first attempt at a classification. Clinically this observer divided his cases into the constitutional and localized varieties, the former denoting the polyarticular types of modern writers, and the latter the monarticular.

With regard to the first group it cannot be doubted that he included within it not only examples of the polyarticular variety to which the application of the term rheumatoid arthritis is coming more and more to be strictly limited, but also cases of osteo-arthritis. Under his monarticular heading he deals almost entirely with osteo-arthritis, citing examples of the affection in connection with most of the larger joints, and in addition referring to instances in which the morbid process was confined to the articulations of the spinal column.

The classification, however, that calls for most attention is that laid down by Charcot,[2] and upon which, indeed, most succeeding ones have been modelled.

He recognizes three main groups :—

1. Progressive chronic articular rheumatism. This type he considered identical with the rheumatic gout of Fuller, the primary asthenic gout of Landré Beauvais, and the nodosities of the joints of Haygarth. Its distinctive features were its progressive character, its tendency to involve many joints, with a special predilection to affect and begin in the smaller articulations, and the severity of the muscular atrophy and contractures.

He further differentiates this group into two subdivisions :

(*a*) Chronic articular rheumatism of rapid evolution, generally arising in young subjects (16 to 30) and especially associated with pregnancy and puerperal conditions, also prone to arise under the influence of emotional causes. The severity of the general symptoms, the rapid invasion of the joints, and the accompanying pyrexia indicate that he was here dealing with the acute and subacute forms of rheumatoid arthritis of modern authors. Pathologically this form was characterized by very slight new bony formations of the shape of elongated needles.

(*b*) Chronic articular rheumatism of slow evolution, chiefly met with in patients from 40 to 60 years of age. Contrary to the former, distinct bony outgrowths are a feature of this class, while there is rarely any febrile reaction. Malpositions due to muscular spasm are absent, the deformities of the joints in this division being due to exostoses.

Here it is important to note that when discussing chronic progressive articular rheumatism as a whole, Charcot points out that during the later stages of the disease certain secondary changes develop which allow of a further differentiation being made into two types, e.g., the "atrophic," and the "œdematous." In the former, atrophy of bones, atrophy of muscles, and wasting of the soft parts are the usual symptoms, in which change, moreover, he says, the skin participates, a condition like scleroderma developing. The recognition of this atrophic type is, as Charcot remarks, due to Vidal, and if we except the absence of any allusion to the early atrophy of cartilage, detection of which is only possible since the advent of skiagraphy, it obviously corresponds exactly to the atrophic or rheumatoid type on which so much stress has been laid by Goldthwait and other writers.

The second, or œdematous type, is characterized by gradual enlargement, especially of the lower limbs, simulating to his mind elephantiasis.

Modern research amply confirms Charcot's clinical experience as regards these two types, the distinctive features of which, if taken together with his definition of subdivision (*a*), give us succinctly the main features of the life history of rheumatoid or atrophic arthritis. On the other hand, as regards his subdivision (*b*) it is, I think, certain that here he was in the main dealing with the opposite condition of osteo-arthritis, and attaching such importance as he obviously did to the presence of bony outgrowths in differentiating his two subdivisions of chronic progressive articular rheumatism, it is somewhat remarkable that he did not relegate the subdivision characterized by slow evolution and osseous formation to the following main group of partial chronic articular rheumatism, which corresponds to our modern hypertrophic or osteo-arthritis.

2. Partial chronic articular rheumatism. In this type the number of joints attacked is limited. Sometimes only one is affected; for example, malum coxæ senilis. Or, on the other hand, several joints may be involved, when the term arthrite sèche was applied.

3. Heberden's nodosities.

Both of these groups are characterized by hypertrophy of bony structures, and are classed as examples of osteo-arthritis.

Of the more modern classifications of arthritis deformans, Osler's[3] is probably the widest known. He enumerates them as follows: (1) Cases with Heberden's nodes; (2) General progressive arthritis, polyarticular, and occurring in both acute and chronic forms; (3) Monarticular forms; (4) Vertebral spondylitis deformans; (5) Arthritis deformans in children.

Recently an attempt has been made by Goldthwait[4] to arrange a classification on pathological lines. He distinguishes three groups: (1) Infectious arthritis; (2) Atrophic arthritis; (3) Hypertrophic arthritis.

As the following definition shows, the term infectious arthritis is here used in no restricted sense. Thus the author says: "The term infectious arthritis is used to designate a joint disease resulting from the presence within the body of some infectious organism, the symptoms being due either to the presence of the organism itself within the joint, or to some toxin produced by that organism in some other part of the body."

This wide and inclusive interpretation of the term infectious arthritis is justified by the fact that many cases of arthritis in which organisms are absent resemble very closely those of ascertained microbic origin—this similarity obtaining not only as regards the character of the local arthritic changes induced—but also in reference to the general aspect of the patients, the mode of onset of the disease, and its clinical course and behaviour under treatment. Inasmuch as the theory of the infective origin of the more acute forms of rheumatoid arthritis rests largely upon such general resemblances to the infective arthritides rather than upon well-established bacteriological data, the author is, from his own point of view, quite consistent in including such cases of rheumatoid arthritis as present these similarities in his group of infectious arthritis—and accordingly we find in this group all examples of Still's disease, and also apparently all cases of arthritis deformans occurring in adults which present similar glandular enlargement. He notes further that in infectious arthritis there is marked leucocytosis in the early stages, with usually a high percentage of hæmoglobin. Later on the leucocytosis disappears, the percentage of hæmoglobin is lowered, and a secondary anæmia develops.

To the second group he applies the term atrophic or rheumatoid arthritis, sharply dividing it off from the previously described type by the following characteristics: its gradual and insidious onset, its essentially chronic course without pyrexia, and certain local peculiarities in the affected joints which, briefly put, are as follows:—

In the early stages we have a typical spindle joint; subsequently the enlarged joint becomes reduced in size even below its normal dimensions, as the result of continuous atrophy of the skin, peri-articular tissues, synovial membrane, cartilage, and bone. This atrophy constitutes, in his opinion, the salient feature of this type. In it, as opposed to infectious arthritis, there is no leucocytosis or secondary anæmia.

The third group needs no further description beyond that it is identical with osteo-arthritis, namely, characterized by "hypertrophy" of bony and cartilaginous structures.

Most authors will agree with Goldthwait that chronic villous arthritis is not to be regarded as a distinct entity, but is a superadded condition common to other joint disorders besides the three types above alluded to, and of these it is more commonly to be met with in the atrophic and hypertrophic than in the infectious variety.

I have thought it necessary to give this detailed account of Goldthwait's work, inasmuch as it is one of the most important and valuable contributions to the sum of our knowledge brought forward in recent years.

Nathan,[5] of New York, another American observer, divides all the diseases associated as rheumatoid arthritis into two great classes: (1) Those which begin acutely, with a more or less definite onset, a rise in temperature, general constitutional reaction, local signs of inflammation, followed by remission or intermission, with perhaps permanent but not progressive joint disability; (2) Those which come on insidiously, with no rise in temperature, slowly progressive course with no intermissions, leading gradually and progressively to complete joint disability. The first he considers inflammatory or infectious in origin, the second trophic. He distinguishes several varieties of the inflammatory form according to their mode of onset and etiology, where such can be ascertained. In one of these sub-groups he places all cases with glandular enlargement, including all examples of "Still's disease," which he states is not confined to children. In another sub-group, where the onset is fulminant, the temperature high, the constitutional reaction severe, and in which visceral complications are common, after the subsidence of the acute attack he believes that two distinct classes can be distinguished according to the joint condition: in the one the bones retain their normal contour, the capsule is much thickened with much contraction of the soft parts; in the second class the articular ends of the bones have lost their normal contour, there is little or no swelling of the soft parts, but occasionally effusion. To the first he gives the name "infectious arthritis," to the second "infectious osteo-arthritis," stating that X rays are very useful in the diagnosis. Finally one of these sub-groups is occupied by those joint affections associated with well-known organisms, such as pneumococcus, gonococcus, and typhoid arthritis, which, according to him, may be arthritic or osteo-arthritic in character. The trophic forms he divides into the synovial and the osseous. The synovial are characterized by effusion, and swelling of the soft parts may arise at any age. The disease usually begins in the peripheral joints, and some cases are associated with marked auto-intoxication. Under the osseous group he distinguishes three types, the first of which is characterized by a persistent advance from joint to joint, and eventually may involve all the articulations; it is attended by well-marked cachexia and emaciation, and ultimately complete crippledom. Marked rarefaction of the articular ends, and sometimes of the shafts, occurs, as evidenced by the X rays, but no bony ankylosis. The second he terms senile arthritis, of which he considers malum coxæ senilis the type. The third form need not detain us, as it deals with the arthropathies of tabes and syringomyelia. In conclusion, the author considers that all these various types of the disease have no direct connection with each other, except in so far as they all affect the joints, each being a definite clinical entity associated with distinct pathological conditions.

In reviewing these last three classifications one cannot help being struck by the growing tendency to limit and define more accurately the use or application of the term rheumatoid arthritis—if not indeed to abolish its use entirely—a wish doubtless born of the sense of confusion which prevailed, and still continues to prevail, as to what did and what did not constitute the rheumatoid condition.

While sympathizing in a measure with those who, like Nathan, advocate its complete excision from the nomenclature, one cannot help feeling that the more moderate suggestion that we should restrict the use of the term, is for the time being, in view of its old associations, the wiser course to adopt.

Nevertheless, I am, in the present state of our knowledge, disinclined to go so far as Goldthwait, who strictly limits the term rheumatoid arthritis to the more chronic forms of the disease, namely, to his atrophic variety, which, as far as I can gather, corresponds very closely to the "chronic rheumatoid arthritis" of English observers. My clinical experience coincides with that of these latter in that there is also an acute variety of the malady to which the term under discussion is also applicable. I make this statement, while fully cognisant of the possibility, or rather certainty, that under the title of acute rheumatoid arthritis cases have been admitted which were undoubtedly instances of infectious arthritis—but if I may judge from the examples given by Goldthwait under the heading of "infectious arthritis"—the converse error is not likely to occur, namely, for cases of acute rheumatoid arthritis to intrude themselves into descriptions of infectious arthritis. To avoid repetition I would refer to the section on "The Diagnosis of Rheumatoid Arthritis," for a detailed discussion of their differentiating clinical characteristics.

Here, because of the great value attached by Goldthwait to X-ray photographs in differentiating his three types, one must draw attention to the fact that even in infectious arthritis the bones may not preserve their normal density, as he maintains, but may exhibit that rarefaction near their articular ends at one time deemed diagnostic of rheumatoid arthritis, but which, as has been shown by Kienbock,[6] may be observed in gonorrhœal arthritis. Moreover, recent work in this direction issuing from the Cambridge School seems to show that from the point of skiagraphy his types show no constant purity, but display a tendency to pass one into the other.

Nathan's classification in a broad sense resembles Goldthwait's inasmuch as he starts with two main groups, an infectious or inflammatory and a trophic, and like the latter observer he includes "Still's disease" in the infective category; it also seems likely that his synovial and osseous types of trophic origin are identical with the atrophic and hypertrophic forms of Goldthwait.

In comparing Goldthwait's with Osler's classification it seems probable that the former's infectious arthritis includes all the latter's acute polyarticular variety, as well as "Still's disease," while his hypertrophic arthritis embraces the monarticular form, the vertebral or spondylitis deformans, Heberden's nodes, and some of the chronic polyarticular, namely those associated with hypertrophic changes in the bone. Those chronic polyarticular cases associated with primary atrophy of the bones correspond to Goldthwait's atrophic arthritis.

It will be noted that both in Goldthwait's and Nathan's classifications rheumatoid arthritis and osteo-arthritis are treated as distinct entities, both clinically and pathologically. More recently Garrod, in Allbutt's *System of Medicine*, Vol. III., 2nd Edition, follows the same mode of division into rheumatoid arthritis and osteo-arthritis, the former approximating to the atrophic, and the latter to the hypertrophic, of Goldthwait. In addition, while he recognizes that minor degrees of spinal involvement are to be met with in the generalized forms of both these diseases, he devotes a special section to spondylitis deformans. Under rheumatoid arthritis he treats of both acute and chronic varieties, making no sharp division between them, while osteo-arthritis he divides into generalized and localized forms.

Pribram[7] suggests the following classification :—

1. Rheumatoid arthritis.
2. Secondary chronic articular rheumatism.
3. Chronic pseudo-rheumatism.
4. Osteo-arthritis deformans.

Hoffa[8], in his exhaustive treatise, recognizes three main groups, namely :—

1. Primary chronic progressive polyarthritis.
2. Secondary chronic articular rheumatism.
3. Osteo-arthritis deformans.

The first-named of these is identical with the rheumatoid arthritis of Pribram, and is also synonymous with the chronic rheumatoid arthritis of the English authors, and the atrophic arthritis of Goldthwait.

The group designated secondary chronic rheumatism calls for particular attention. Clinically, this group differs from that of primary chronic progressive polyarthritis only in the fact that it begins more acutely, and is said either to arise out of, or have an undoubted connection with, a previous attack of acute articular rheumatism. It is significant that Hoffa confesses that he is unable to distinguish between the anatomical lesions produced in this variety and those characteristic of primary chronic progressive polyarthritis. In the latter condition, the joint-affection arises in an insidious manner, pursuing an almost apyrexial course, and eventually presenting the typical appearance of chronic rheumatoid arthritis of English authors. When we bear in mind the extreme difficulty in differentiating at their onset the more acute forms of rheumatoid arthritis from acute articular rheumatism, it seems to me justifiable to assume that, in secondary chronic articular rheumatism, we are dealing with rheumatoid arthritis of an acute onset.

Taken together, this latter group, and that of primary chronic progressive polyarthritis, seem to me to represent the acute and chronic rheumatoid arthritis of Garrod and other English authors.

Both Hoffa and Pribram draw a sharp distinction between the above two groups and osteo-arthritis deformans; while it may be added that the variety termed by Pribram chronic pseudo-rheumatism is identical with the infectious arthritis of Hoffa, Goldthwait, and others.

In conclusion, as regards the scheme of classification that will be adopted in this work, I shall take as my basis that there are two main groups of arthritis deformans: (1) Rheumatoid arthritis; (2) Osteo-arthritis. Under the first-named heading will be included not only the atrophic arthritis of Goldthwait, but also the more acute forms of rheumatoid arthritis, showing glandular and occasionally splenic enlargement, all of which, including "Still's disease," Goldthwait, as before stated, would relegate to his group of infectious arthritis. On purely clinical grounds, because of the existence of endless transitional forms, it is impossible to differentiate clearly, on the one hand, between the more acute and the chronic forms of rheumatoid arthritis, while on the other hand, on the same basis, and for the same reasons, it is difficult if not impossible to draw a sharp dividing line between the former group and that of the infective arthritides.

It is to be hoped that the exact lines of fusion will before long be determined by fresh bacteriological data. Pending that, I prefer to adhere to this wider interpretation of the term rheumatoid arthritis than to adopt Goldthwait's definition of that affection.

Pathologically, in this group, the morbid changes occur primarily in the softer structures, the peri-articular and synovial tissues undergoing thickening, with hypertrophy of the synovial fringes, while ultimately a process of atrophy, involving all the constituent elements of the joints, takes place, including the bony and cartilaginous, as well as the softer, tissues.

In osteo-arthritis the primary changes take place in the cartilaginous and bony structures, which undergo enlargement or hypertrophy, with the formation of osteophytic outgrowths, thus contrasting sharply with the essential atrophic process characteristic of the first-named group, in which, if new bony formation occur, it takes place secondarily, and is inconsiderable in amount.

The osteo-arthritic group, for the better convenience of description, will be dealt with under two headings—the generalized and the localized,--while special sections will be devoted to arthritis deformans as it occurs in children, and to spondylitis deformans.

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Section II.

RHEUMATOID ARTHRITIS.

SECTION II.—RHEUMATOID ARTHRITIS.

CHAPTER III.

ETIOLOGY.

THE etiology of rheumatoid arthritis is extremely obscure. Speaking in the exact sense, nothing is accurately known as to its causation, and it must be frankly admitted that for the present our attempts to assign an origin for the disease consist simply in the enumeration of a series of antecedent or pre-existing diseases separated by a more or less variable interval from the subsequent rheumatoid arthritis into relationship with which they are presumed to enter.

To prove that any intimate connection exists between rheumatoid arthritis and these very diverse factors is, in the present state of our knowledge, extremely difficult, and would necessitate, for one thing, the establishment of the existence of a series of symptoms linking up the condition with its assumed etiological factors. So much controversy obtains as to the existence or non-existence of premonitory symptoms in the period of invasion, so to speak, of rheumatoid arthritis, that I have endeavoured, so far as it lies in my power, to pay particular attention to this period of the disease ; and, as a result, I have no doubt that such do exist. Moreover, they occur so frequently, and are so constant in their characteristics, that I consider they will yet be found to throw considerable light on this vexed question of the exact etiology of the affection. Into a detailed description of these inter-connecting symptoms and their relationship to the subsequent rheumatoid arthritis I do not propose to enter here, seeing that they will be dealt with in a later chapter. Inasmuch as most authors have dealt with the etiology from the point of view of arthritis deformans as a whole, and have not attempted to differentiate that of rheumatoid arthritis from osteo-arthritis, we can get but little help on this point at any rate from the older authors. Goldthwait,[1] who advocates the distinctness of the two affections, when discussing this question of the causation of rheumatoid arthritis, states frankly, "The etiology of the condition is not known."

Proceeding now to consider in detail the various factors that appeared to enter into relationship with those examples of the disease that came under my own observation: To begin with, the affection shows undoubtedly an overwhelming predilection for attacking the female sex. Taking 240 consecutive cases out of my series of rheumatoid patients, there were 39 males to 201 females: a proportion of 1 to 5, or 16·25 per cent and 83·75 per cent respectively.

In another series of 150 examples particular attention was paid to the age at which the onset of the disease took place; and its varying incidence in the several decades will be shown by the following table:—

	0–10	10–15	15–20	20–30	30–40	40–50	50–60	60–70	Total
Females	2	6	24	37	24	26	9	0	128
Males	0	1	1	6	11	3	0	0	22

I shall here take the liberty of comparing the above table with the following one recorded by Garrod,[2] who, like myself, has taken care to include only cases exhibiting the characteristic features of rheumatoid arthritis:—

	0–10	10–15	15–20	20–30	30–40	40–50	50–60	60–70	Total
Females	1	3	3	22	28	14	5	1	77
Males	0	0	0	7	6	3	7	0	23

It will be seen that in my series the period of greatest incidence in both the female and the male sex lay between the ages of twenty and forty, which confirms the results previously deduced by Garrod from his table.

Again, McCrae,[3] in a series of ninety-two cases, which he defines as being of the polyarticular type in which the changes were largely periarticular, thus permitting of legitimate comparison with Dr. Garrod's and my own series, gives the ages of onset in the following table:—

Ages of onset.	No.
1–10	11
11–20	17
21–30	18
31–40	22
41–50	10
51–60	11
61–70	2
71–80	1

Here again it will be seen from the above figures that the age of greatest incidence closely approximates to that indicated by Dr. Garrod's series and my own.

Previous History.—Taking a series of 240 cases of purely rheumatoid type, the antecedent diseases from which the patients had suffered occurred in the following proportions :—

Of acute specific disorders influenza was the most frequent, a history of it being forthcoming in 63 cases ; of the remaining zymotic diseases, a history of measles was obtained in 18, of scarlet fever in 11, diphtheria in 9, small-pox in 5, pneumonia in 5, erysipelas in 3, enteric fever in 2, chicken-pox in 2, ague in 1.

Nine had been the subjects of gonorrhœa ; in 41 there was a previous history of one or several attacks of a polyarthritic nature, variously described as "rheumatic fever," "acute rheumatism," or "rheumatism." Twenty-six had been subject to oft-recurring sore-throats, defined as "quinsy," "tonsillitis," and so forth. In view of the disputed relationship of acute rheumatism to rheumatoid arthritis it is interesting to note that four had suffered from chorea.

Next in order of frequency come disorders of the gastro-intestinal tract : in 8 there existed a condition of oral sepsis ; in 7 a condition of gastric ulcer, attended in most instances by hæmatemesis ; while in 21 chronic indigestion had been a prominent feature of their previous history.

Genito-urinary diseases come next : one patient suffered from nephritis ; the disease in 16 appeared to arise in quick sequence to a miscarriage ; in 1 the joint swellings appeared during pregnancy ; while 3 had suffered from "white leg."

The onset of 5 was associated with the menopause ; in 8 a history of previous amenorrhœa was forthcoming ; menorrhagia in 9 ; while 35 had suffered for long periods with chronic leucorrhœa, and 28 from anæmia previous to the disease. Of the remainder, 8 had suffered from pleurisy, 8 from otorrhœa, 2 from nasal catarrh, 4 from asthma, 8 from migraine, 1 from epilepsy, 7 from mental shock or worry. In only 1 was there a history of alcoholic excess, and in 1 a history of trauma.

Proceeding now to discuss the etiological relationship of these scattered factors to rheumatoid arthritis :

With regard to the acute infectious disorders the most noticeable feature is the frequency with which influenza figures in the past history of rheumatoid patients. That an unusually obstinate form of arthritis may follow influenza is now well known. Both Howard Marsh[4] and Wallis[5] have called attention to its occurrence, the former noting that it was characterized by a slow course, and attended by œdema, pain, and muscular wasting. Having regard to the frequency with which a history of influenza is forthcoming in rheumatoid patients, the question naturally arises as to whether there is any causal relationship between the two affections. In some of the above series the initial onset of the disease seemed directly related to the preceding attack of influenza. Moreover, as Poynton has observed, both in the subjects of rheumatism and those of rheumatoid arthritis a severe recrudescence of their respective joint troubles has often been observed to follow an attack of this disorder.*

Again, we have in influenza an infection whose toxins evince an undoubted affinity for the nerve centres, one whose effects are extremely lasting. Indeed, so constant are these nervous sequelæ in their appearance, that in the case of a febrile attack of doubtful nature, a very reliable indication of its influenzal character is afforded us by the neurasthenic and depressed condition that so often ensues. Given, then, a condition of arthritis supervening after influenza and accompanied by collateral features of nervous type, we have undoubtedly a close approximation to what obtains in rheumatoid arthritis.

If it could be proved that Pfeiffer's bacillus was present in the joints in rheumatoid arthritis, the associated trophic and vasomotor disturbances would be readily explained by coincident invasion of the nerve centres by its toxins.

Hitherto, unfortunately, there is no evidence that direct infection of the joints by this organism does occur. Therefore, while conceding the possibility of a causal relationship, one is led to infer that the arthritic changes are produced by toxins derived from some remote site—as, for example, the gastro-enteric tract, direct infection of which by this organism sometimes occurs.

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*Poynton, discussing the possible etiological relationship of influenza to rheumatoid arthritis, refers to an interesting case in this connection reported by L. Dudgeon and Adams.

The subject was an infant aged ten months, who developed arthritis of the left elbow and right hip joint. After an illness of two months the patient succumbed, and from a purulent exudation in the right hip joint these observers isolated an organism in pure culture which was apparently identical with the bacillus of influenza.

The same organism was obtained from the affected elbow joint as well as from the spleen. (*Lancet*, Sept. 7th, 1907.)

It is also noteworthy that other specific infectious disorders known to be associated not infrequently with joint affections were also etiological factors, such as scarlet fever. In two of my cases the patients had experienced an attack of so-called scarlatinal rheumatism. Strangeways[7] indeed states that rheumatoid arthritis may follow closely on scarlatina, and cites three instances in which the joint troubles actually developed during the course of the disease. While the history of measles was only obtainable in eighteen of my cases, it occurred in no less than sixty-eight times in McCrae's series; on the other hand, only thirteen of his patients had suffered from influenza.

The fact that only nine of my series, or 3·75 per cent, gave a history of gonorrhœa, is in striking contrast with Stewart of Montreal's published statistics of 40 per cent. McCrae, however, fixed the frequency in his series at 13 per cent.

We have before alluded to the fact that forty-one of the cases when they came under observation gave a history of an antecedent attack of polyarthritis. In many of these individuals several illnesses of this nature had been experienced; and in some instances the intervening periods were to be measured by years, in others by months. Each fresh outbreak was, as a rule, associated with some intercurrent disorder, and very frequently an exacerbation was directly referable to an attack of influenza, quinsy, or gastric disorder.

It is worthy of note that complete recovery from the earlier attacks of polyarthritis took place in some instances.

McCrae also has noted these recurring attacks of polyarthritis as a striking feature of the previous histories of rheumatoid patients; and, like him, I am inclined to believe that in the vast majority of instances the condition *ab initio* was one of rheumatoid arthritis. An exception, however, must be made in those few cases in which the past history revealed in addition the occurrence of chorea. This disease is undoubtedly a purely rheumatic manifestation, and it seems almost certain that the preceding acute polyarthritis was of the same nature. This sequence of events—acute rheumatism, chorea, rheumatoid arthritis—as far as it goes, does apparently favour the contention of some that an obscure causal relationship exists between acute rheumatism and rheumatoid arthritis.

Similar inferences have been drawn from the occasional appearance of subcutaneous fibroid nodules and signs of cardiac involvement in the subjects of rheumatoid arthritis.

This sporadic manifestation of recognized rheumatic lesions in rheumatoid patients is extremely difficult to account for, especially if the disease from its onset has pursued an acute course.

That the gonococcus may give rise to a variety of arthritic changes, including those of rheumatoid arthritis, is generally admitted; and one cannot exclude the possibility that the organism responsible for acute rheumatism may possess similar capabilities, the diversity in the tissue-reactions being determined by peculiarities in the soil.

Another hypothesis is open to us, namely, that the particular virus of rheumatoid arthritis has succeeded in establishing itself on a soil which, by reason of a previous attack of acute rheumatism, is predisposed to further arthritic developments.

By this latter view no direct causal relationship on the part of acute rheumatism to rheumatoid arthritis is assumed, and it therefore does not conflict with the modern tendency to regard these two diseases as essentially distinct from one another, an opinion which it is to be hoped will soon be ratified by some decisive bacteriological distinction.

The previous occurrence of repeated attacks of tonsillitis in some rheumatoid patients is an important feature in view of the tendency nowadays to regard the tonsils as a favourite site for the entrance of infectious organisms, not only in acute rheumatism, but also in the chronic non-tuberculous forms of arthritis. Bloodgood,[6] when discussing the infective origin of acute and chronic forms of arthritis, states, "The tonsils stand first as the portal of entrance in non-tuberculous arthritides except the gonorrhœal." From my own experience, I am inclined to believe that the etiological importance of throat infections in rheumatoid arthritis is underestimated; and, like Bannatyne, I have in several instances traced the onset of the affection to chronic catarrhal tonsillitis.

Among the many diseases that may pre-exist, or run their course side by side with rheumatoid arthritis, are those pertaining to the alimentary canal. Oral sepsis, chronic dyspepsia, gastric ulcer, dilatation of the stomach, mucous colitis, ulceration of the intestines, and chronic diarrhœa have all been cited in this connection.

Some years ago I published a series of cases, the course and symptomatology of which seem to point to some obscure relationship between such disorders and rheumatoid arthritis, and with the object of demonstrating the nature of this association I will here insert a few illustrative cases.

M. A., female, æt. 23; single; domestic servant. When a child she had measles, and occasionally sore throat. Since eighteen years of age she had suffered with anæmia. The anæmia became very much worse during her twentieth year. She could hardly follow her occupation as a servant, and had to sit half way on the stairs, owing to shortness of breath. Amenorrhœa for three months at a time was present. Constipation was marked.

During this period she suffered severely with indigestion. Pain shooting through to the back, accompanied by severe vomiting, was present. The vomiting was very troublesome for three months. About this time she noticed that her index, middle, and ring fingers used to go "dead white" from time to time. The change in appearance was accompanied by "cramp" of same fingers, which used to become fixed in a position of semi-flexion. These seizures were most marked in the morning, and were provoked by exposure to cold. They recurred daily, sometimes more often. During the same period she noticed her feet used to go blue and cold. Swelling of the joints of fingers and puffiness of dorsum of feet gradually supervened. Other joints, right elbow, and temporo-maxillary, were subsequently involved. The vomiting, etc., above referred to, continued off and on for two years, and is still present, though less severe and less frequent, and any increase in severity of gastric symptoms is followed by an exacerbation of joint troubles.

She was admitted to the Mineral Water Hospital for relief of her joint condition, which presented the usual characteristics of rheumatoid arthritis. There was, in addition, much muscular wasting; also fine tremor of limbs. The deep reflexes in upper and lower extremities were increased, likewise the jaw reflex; and tapping of nerve trunks produced contraction of the muscles supplied by them. The vasomotor phenomena above referred to were well marked, and recurred from time to time. Her teeth were extensively carious; her bowels constipated. Nothing abnormal was found in heart or lungs. Her urine contained no albumin.

E. G., æt. 36; single.

Personal History.—Had scarlet fever and measles in early childhood. Had chorea when fourteen years old. Rheumatic fever at twenty-one years of age; and was then confined to bed for over six months. Seven years ago she became anæmic. This was followed by severe indigestion, accompanied by vomiting, the latter at one time being so bad as to necessitate rectal feeding.

During this period of gastric trouble she had several abscesses around her teeth, all of which, save two, were extracted. Ever since this illness she has suffered off and on with indigestion, vomiting, and diarrhœa. In addition, she was troubled greatly with "cramps." These were very painful, and affected her arms, legs, also muscles of neck. These muscular spasms when they occurred in the legs sometimes caused dorsiflexion of the ankle, at other times plantar flexion, preventing any attempt at walking. At other periods, the plantar muscles were affected. causing strained positions of toes. Her hands during the paroxysms assumed a position of semi-flexion, and when the neck muscles were involved the head was rotated and flexed to one side. Coincidently she suffered with recurrent asphyxias of her extremities, especially the hands. Last September twelve months her gastric symptoms—vomiting, diarrhœa—became very much aggravated, and simultaneously the muscular cramps and vasomotor phenomena. Her joints now gradually began to swell—the fingers, left elbow, ankles, and shoulders—for treatment of which she was sent to Bath. On admission she was seen to be suffering from rheumatoid arthritis, with fusiform enlargement of the finger joints, much muscular wasting, also fine tremor of the limbs. Her deep reflexes were very brisk, both in the upper and the lower limbs, and Chvostek's symptom was easily educible over both ulnar nerve trunks. During her stay in hospital she suffered from vomiting and diarrhœa, and her joints became worse coincidently. Amelioration of the gastric symptoms was followed by a diminution in her arthritic troubles. Heart and lungs were apparently normal. Pulse-rate was 80 per minute.

A. E., æt. 24; female; single.

Personal History.—Measles; sore throats. When nineteen years of age became very anæmic. This condition of anæmia recurred three times. About two years ago she began to suffer with indigestion; also with abscesses around her teeth. She had eleven teeth extracted; but no false ones were inserted. Her indigestion still continued. Two months afterwards she noticed her feet became very "stiff," with pain in the soles; and she suffered with chilblains on toes and heels. Her hands at the same time used to go "blue," and gradually swelling of her joints ensued, and she now presents the characteristic joint swellings of rheumatoid arthritis, involving the temporo-maxillary joint as well as those of the limbs.

The sequence of events in the foregoing clinical accounts seems to indicate some causal relationship between the primary gastro-intestinal disorders and the subsequently developed rheumatoid arthritis.

The existence of interconnecting symptoms linking up the gastric and arthritic troubles, viz., muscular cramps and vaso-motor phenomena, will have been noted, and incidentally one may comment on the resemblance of the muscular spasms to those met with in tetany, a condition not infrequently ascribed to toxic absorption from the alimentary canal.

The vasomotor phenomena likewise have been attributed to poisons generated at the same site.

Armstrong,[7] of Buxton, some years ago suggested that such Raynaud-like conditions were probably the effect of circulating toxins of gastro-enteric origin. Cammidge[8] also recently observed that "coldness" of the extremities was often complained of by the subjects of intestinal toxæmia, and not infrequently was accompanied by enlargement of the metatarsal joints. Again, Sir William Bennett has noted the occurrence of synovial effusions in patients with gastric derangement, and was inclined to believe that some causal relationship existed, inasmuch as successful treatment of the arthritic trouble supervened only when the dyspeptic conditions were corrected.

Post-dysenteric arthritis, of which I saw many examples after the late Boer war, is also usually attributed to circulating toxins derived from the intestinal tract.

The question now arises as to whether the joint swellings of rheumatoid arthritis are not a late manifestation of a long-continued gastro-intestinal toxæmia, the earliest indications of which are the muscular cramps and vasomotor phenomena before referred to, and which, as we have seen, preceded and, so to speak, led up to the joint involvement.

Personally, I am of opinion that in a greater percentage of rheumatoid patients than my series indicates, toxic absorption from the alimentary canal plays an important etiological rôle. Strangeways,[9] in two separate series of rheumatoid patients, consisting each of one hundred examples, found that in one 39 per cent and in the other 35 per cent gave a definite history of dyspepsia.

Such a conception of its nature is tantamount to ascribing the affection to an auto-toxæmia.

The theory of auto-intoxication has of late, however, fallen into so marked disfavour that one is a little loth to invoke its aid to explain the origin of rheumatoid arthritis. But while admitting the salutary effect of scepticism in setting a limit to conjectures in this direction, one cannot but feel that wholesale repudiation of the possibility of auto-intoxication as a cause of disease is as much to be deprecated as a too credulous acceptance of it. *In medio tutissimus ibis.*

A recent writer, Tubby,[10] discussing the etiology of rheumatoid arthritis, states, "The weight of evidence is in favour of the disease being an auto-toxæmia."

It will therefore not be out of place to consider what evidence there is that chronic poisoning from gastro-intestinal products is of pathological import.

Much of the light recently thrown upon this obscure and intricate subject we owe to the researches of Herter,[11] who from clinical and experimental observations concludes that absorption of the chemical products of intestinal fermentation and putrefaction, if long continued, does exert a deleterious effect upon the organism.

By putrefaction is here understood the cleavage of proteid and allied substances, as opposed to the decomposition of carbohydrates and fatty substances through fermentation.

These diverse processes are, however, intimately associated in the digestive tube, excessive fermentation almost invariably inducing abnormal putrefaction.

Now, while it is impossible to positively affirm that no toxic effects are produced by the products of fermentation, it has been definitely established that those due to putrefaction are much more harmful to the organism by reason of their nitrogen- and sulphur-containing compounds.

The chief substances formed during intestinal putrefaction are the aromatic bodies, ammonia, indol, skatol, and phenol, which, however, save in the presence of excessive putrefaction, are not formed in any large amount in the bowel. By the first-named product, ammonia, it is probable that but slight toxic effects are produced, inasmuch as in the liver and elsewhere it unites with carbon dioxide to form urea, and is thus disposed of.

As regards indol and skatol, it is, however, by no means so certain that we can dismiss their poisonous effects as trivial.

In order to estimate the toxic effects, if any, produced by absorption of these bodies, Herter induced some persons to take considerable amounts of pure indol crystals, and concluded that though only moderately toxic to man, yet "The continual absorption of enough indol to give a strong reaction for indican in the urine is capable of producing neurasthenic symptoms." Similarly, he noted that skatol, if administered to monkeys either orally or subcutaneously, was followed by the appearance of some substance in the urine giving the Ehrlich reaction, and also that 0·1 gram of skatol given to man heightened the Ehrlich reaction, while at the same time general toxic effects resembling those of indol were produced.

Passing now to the clinical side, we know that in certain cases of excessive intestinal putrefaction in children, attended by abdominal distention and stunted growth, a large amount of indican and phenol is contained in the urine. These children tire very rapidly, and are slow in learning to walk; and it has been suggested that the readily-induced fatigue and muscular weakness are due in part to the action of phenol and indol.

Again, excessive indicanuria has been observed in neurasthenia, anæmias, chronic gastritis, and in intestinal disorders associated with diminished secretion of bile and pancreatic juice.

But more interesting in view of our subject is Herter's statement that similar indications of abnormal intestinal putrefaction were regularly found by him to be present in all cases of early rheumatoid arthritis. Subsequently this observation was confirmed by Dr. Helen Baldwin working in Herter's laboratory. This observer's results were based upon a study of twenty-one examples of rheumatoid arthritis, in fifteen of whom the disease was progressing, while in the remainder the condition was stationary or improving.

In three of the former cases the stomach contents were examined; and in all of them, after a test meal, there was found to be an absence of free HCl, though there were no other symptoms of gastric disturbance.

In addition, in all but two examples, and those of a mild type, there was noted, either an excess of aromatic sulphates in the urine, or else the presence of indican. On the contrary, in those patients with stationary or improving lesions analysis showed the urine to be normal. Dr. Baldwin[12] considered that the regularity with which the changes in the urine occurred seemed to suggest that some of the nutritional disturbances in rheumatoid arthritis were due to intestinal putrefaction.

Many other writers, among them Armstrong, have also remarked on the frequency with which an excess of aromatic sulphates is to be noted in the urine of rheumatoid patients.

It is necessary here to digress in order to discuss, firstly, the microbic agents responsible for the induction of intestinal putrefaction, and, secondly, those pathological conditions which apparently favour and promote these abnormal bacterial activities.

Putrefactive changes in the bowel, unlike those due to fermentation, are mainly achieved through the action of anaerobic bacteria. Now it has been shown by the researches of Grigoroff, Combe, and others that the organisms met with in the normal intestine are either aerobes or facultative anaerobes, strict anaerobes, on the other hand, being present in extremely small numbers. If, however, abnormal putrefaction is present, then a notable diminution takes place in the aerobic organisms which check, and a corresponding increase in the anaerobes which promote and favour, putrefaction.

Herter distinguishes three types of chronic excessive intestinal putrefaction :—

1. The indolic type, due to invasion of the small intestine by members of the *B. coli* group, and characterized by marked indicanuria, with usually stools of an alkaline reaction.

2. The saccharo-butyric type, in which the large intestine and lower ileum are the site of the putrefactive process. This variety is due to the activity of strict anaerobes, largely *B. aerogenes capsulatus*, *B. putrificus*, and, according to some, the bacillus of malignant œdema. The stools in this form may be acid in reaction.

3. The combined indolic or saccharo-butyric type.

In this, the commonest variety of intestinal putrefaction, the bowel swarms with putrefactive anaerobes, and a high degree of indicanuria prevails.

The morbid symptoms that supervene in the subjects of long-continued intestinal toxæmia vary widely ; and Herter believes that this diversity in the clinical phenomena of such intoxications is attributable, not to differences in the poisons generated, but to peculiarities in the cellular reactions of different individuals. Such variations in vulnerability on the part of the tissues determine whether the blood or the nervous system shall bear the brunt of the toxic action.

All grades of anæmia, approaching even the picture of the pernicious type, may ensue ; and on the side of the nervous system striking degrees of muscular asthenia and mental instability may be experienced.

As might be expected from the great frequency with which one or other of these varieties of intestinal derangement is to be met with, nature has endowed the human body with means of defence against the pernicious effects of the chemical products continually being produced in the gastro-intestinal tract as a result of microbic activity.

Briefly considered, these self-protecting powers reside in the acidity of the gastric juice, the selective action of the intestinal epithelium, the detoxicating functions of the liver, and the assumed antitoxic capabilities of the ductless glands.

Obviously, no consideration of the problem presented by intestinal toxæmia would be complete, if it failed to take cognizance of those pathological conditions which tend to impair the efficiency of these defensive agencies. Herter believes that the chief causes are: (*a*) Diminished secretion of free hydrochloric acid; (*b*) Diminished secretion of the intestinal digestive juices; (*c*) The excessive use of proteid food, especially meat.

Absence or insufficiency of free HCl in the gastric juice favours the growth of anaerobic organisms which may have gained an entrance to the stomach, and incidentally increases the chance of such bacteria reaching the intestine, where they promote putrefaction. This diminished acidity also facilitates putrefaction, by permitting the passage into the intestine of ill-digested proteid substances, which in this condition are much more prone to undergo putrefaction.

Now, it will have been noted that in three of Dr. Baldwin's examples there was an absence of free hydrochloric acid; and Herter believes that the excessive indicanuria noted so often in rheumatoid arthritis is in part due to this deficiency, or absence, of free HCl. It is possible also that the condition of oral sepsis, so frequently found in rheumatoid arthritis, plays a part in breaking down this defensive action on the part of the gastric juice.

It seems probable that dental caries is due to the action of anaerobic organisms, which find a suitable nidus for their growth in particles of food lodged in or between the teeth. Now, while the gastric juice, when normally acid, is quite capable of dealing with moderate quantities of pathogenic bacteria, it is only too likely that in patients with oral sepsis a greater number of bacteria are swallowed with food than can be satisfactorily coped with. Mr. J. Lewin Payne,[13] dental surgeon to Guy's Hospital, has noted the frequency with which patients with carious teeth show signs of rheumatoid arthritis. Pyorrhœa alveolaris has also been observed in the same connection; and it is easy to see how, in such cases, pyogenic organisms must constantly be carried into the stomach with food.

Macalister,[14] of Liverpool, assigns great importance to the nasal passages as a favourable site for infection in rheumatoid arthritis; and Herschell,[15] when discussing the causes of intestinal putrefaction, regards post-nasal catarrh as an etiological factor of some importance. Thus he points out that the subjects are continually and automatically swallowing mucus, in varying amounts, containing noxious organisms, most frequently the bacillus of Friedlander; and this, moreover, goes on during the night, when free HCl is absent from the stomach.

It seems probable, therefore, that the etiological relationship of chronic infections of the mouth, throat, and accessory cavities to rheumatoid arthritis finds here its most reasonable explanation; namely, that in their presence the gastro-intestinal tract is not unlikely to become infected, thereby inducing putrefactive changes in its contents.

Passing now to consider the relationship of diminished secretion of the intestinal digestive juices to putrefactive processes: catarrhal affections of the stomach and small intestine constitute the most common cause of reduction in the secretion of bile and pancreatic juice. As Cammidge recently pointed out, impairment of the digestive powers of the pancreas is followed by far-reaching consequences. Food stuffs undergo abnormal changes, are less easily absorbed, and, simultaneously, chemical products are formed which irritate the intestinal mucosa.

The baneful effects of these digestive disturbances do not cease here, for as a result of the unusual nature and altered reaction of the intestinal contents the flora in the bowel undergo changes. Thus, organisms normally present only in a minority in the small intestine may find the altered medium more favourable to their growth, and consequently multiply, while others constantly present in the lower bowel may migrate upwards to the ileum and duodenum, and even infect the pancreatic and biliary passages.

As a consequence of the deficient pancreatic ferments, proteins are imperfectly digested, and putrefaction under microbic action is favoured, while the digestion of carbohydrates is impaired, organic acids are formed, and gases in large amounts liberated. Ultimately, owing to absorption of these irritating products, a condition of chronic toxæmia ensues.

One of the most important observations made by Cammidge, in his study of "Intestinal Indigestion," is the statement that such disorders are occasionally to be traced to a preceding attack of influenza or typhoid fever. When such occurs, pancreatitis and cholangitis not infrequently ensue, leading, more particularly the former, to impaired digestion with all its remote consequences.

As we saw when dealing with the etiological relationship of the specific infectious diseases to rheumatoid arthritis, influenza figured very prominently as an antecedent. In addition, we noted that in such cases the influenza bacillus had not been detected in the joint cavities, its absence leading one to infer that the toxins responsible were formed at some remote site. Now it seems possible—in the light of Cammidge's researches—that infection of the intestinal tract and the related biliary and pancreatic passages by Pfeiffer's bacillus may, through the ensuing intestinal toxæmia, give rise to the condition of rheumatoid arthritis.

I am led to this conclusion because, in cases of rheumatoid arthritis preceded by influenza, the same interconnecting symptoms, muscular cramps and vasomotor phenomena, were found linking up the two conditions in a manner exactly similar to what was observed in cases of rheumatoid arthritis following gastro-intestinal disorders. The researches of A. O. Kelly and others have demonstrated that influenzal, pneumococcic and pyogenic infection of the biliary passage occur much more frequently than was thought, and in some instances the pathway of entry is *via* the diverticulum of Vater.

At the present time I have under my care a woman, the subject of rheumatoid arthritis, who for many years has been a sufferer from dyspepsia and biliary colic, for which she recently had to undergo cholecystotomy. Although the co-existence of these two conditions may be only a coincidence, it is conceivable that the intestinal and biliary affection formed the starting-point of a chronic toxæmia.

As we now know, these masked infections of the biliary and pancreatic ducts may be very readily overlooked, the local symptoms pointing to such conditions being often absent, or extremely slight—much less obtrusive than the remoter consequences of the toxæmia they induce.

Here it seems opportune to refer to a recent contribution by Dr. Katherine S. Clark[16] dealing with the etiology and pathology of rheumatoid arthritis. This observer, in reviewing the clinical features of her series of cases, emphasises the invariable association of gastro-intestinal derangements with the condition. In three of her cases which came to autopsy definite pathological changes were found in the liver, pancreas, and other viscera.

In the liver, in two of the cases, there was an increase of connective tissue in the portal tract, with thickening of the walls of ducts and vessels, this fibrosis being very extensive in one case.

The pathological changes in the pancreas were even more pronounced, consisting of marked overgrowth of connective tissue throughout the organ, with degeneration of the parenchyma, hyaline thickening of vessels and ducts, with proliferation of their lining epithelium.

In the kidney also pathological changes were evident—thickening of the arterial walls and hyaline degeneration. The glomeruli in all cases showed marked hyaline changes, whilst throughout the organ were scattered patches of fibrosis. Now, similar changes have been noted in the subjects of chronic excessive intestinal putrefaction; thus, Dutton Steele notes the occurrence occasionally of chronic parenchymatous changes in the liver and kidney due to the persistent action of poisons formed in the alimentary tract.

The foregoing pathological changes noted in the viscera of rheumatoid patients are of much interest, especially those relating to the liver and pancreas.

As we pointed out when dealing with the defensive powers of the organism, the liver exercises a detoxicating function; or, to use Herter's metaphor, it may be compared to a sentry constantly on guard to screen the nervous system from the injurious effects of toxic substances formed in the intestinal canal. Thus, for example, when as a result of proteid putrefaction indol is formed, it is carried by the portal stream to the liver, there undergoing oxidation and transformation into a product which may safely be allowed access to the general circulation.

Obviously it follows that any impairment of the functional integrity of the hepatic cells entails enhanced risk of such bodies as indol remaining unoxidized, and, reaching the general blood-stream in this form, they may exert the injurious effects upon the nervous system before alluded to.

Combe,[17] in his work on "Intestinal Auto-intoxication," lays stress upon this protective action of the liver, which he terms the second line of defence; while, in addition, he recognizes a third barrier constituted by the ductless glands. It is necessary to refer briefly to the last-named organs, because Lancereaux,[18] Macalister, and others believe that an obscure etiological relationship obtains between disorders of the thyroid secretion and some cases of rheumatoid arthritis.

Vincent,[19] of Val de Grâce, has noted that in the course of various infectious diseases the thyroid gland may undergo more or less marked hypertrophy. Such enlargement may remain permanently, giving rise to symptoms of hyperthyroidism, or even Graves's disease; or, on the other hand, the gland may atrophy from functional exhaustion.

In the latter instance symptoms of thyroid insufficiency arise, among the effects of which are to be included the condition of "chronic articular rheumatism."

Enlargement of the thyroid gland has been frequently observed in rheumatoid patients; and I have myself noted some instances in which the symptoms of Graves's disease and rheumatoid arthritis coexisted. In one of my cases, a woman, the subject of exophthalmic goitre, the thyroid enlargement underwent recession, and a condition of rheumatoid arthritis quickly developed.

The striking improvement that has followed the administration of thyroid gland in some cases of rheumatoid arthritis tends to support this hypothesis. It may also be mentioned that in a female, the subject of the atrophic variety of spondylitis deformans (spondylose rhizomélique) Rue and Babboneix[20] found at autopsy extensive changes in, not only the thyroid, but also the pituitary and the pancreatic glands, as well as the ovaries. They attributed the extensive degeneration of these structures to the cumulative toxic effects of a rapid succession of acute and infectious disorders from which the patient had suffered during childhood.

Among the many theories advanced to explain the origin of Graves's disease has been the hypothesis that it is due to toxins absorbed from the alimentary tract. In a case under my own observation the primary gastro-intestinal disorder was followed by the development of rheumatoid arthritis and Graves's disease; and it appeared to me that the Graves's syndrome was a further manifestation of the chronic toxæmia which had presumably caused the rheumatoid arthritis.

In reviewing this attempt to explain the etiological relationship of gastro-intestinal disorders to rheumatoid arthritis, one is conscious that much remains to be done before such a theory of its causation can be considered as established. Still, despite the mystery that envelops this question of intestinal auto-intoxication, clinical experience distinctly suggests that such a condition exists.

The most cogent facts in favour of this theory in the case of rheumatoid arthritis are: Firstly, the great frequency with which gastro-intestinal disorders precede, or accompany, the joint affection; secondly, the existence of interconnecting symptoms; thirdly, the presence of conditions favourable to intestinal putrefaction, such as oral sepsis, deficiency of free HCl; fourthly, evidences of intestinal putrefaction, as found in the urine; lastly, the beneficial effects of treatment having for its object the diminution of intestinal putrefaction.

We pass now to consider the relationship of uterine disorders to rheumatoid arthritis. Its association with pregnancy, puerperal conditions, and the menopause, was pointed out by Charcot;[21] and nearly every writer since has confirmed him, in that women at such times are very liable to develop the disease. The onset actually during pregnancy has been recorded in several instances; and I have myself known exacerbations of the disease to occur during gestation. More commonly the affection arises some weeks or months after delivery, contrasting in this respect with the so-called puerperal forms of arthritis. The same tardy appearance characterizes cases following miscarriage.

It may be a coincidence, but I have noted in these puerperal conditions that the rheumatoid affection is often preceded by neuralgias of various sorts, more particularly of cervico-occipital distribution, and attended by transient stiffness of the neck. Such symptoms are often ascribed by patients to exposure of their neck and shoulders while nursing fretful infants at night.

In other instances muscular cramps are prominent antecedent symptoms; and one is reminded of Trousseau's observation on the tetany of nursing women. Gastro-intestinal derangements are not uncommon during pregnancy and the puerperium, and gestation is also not infrequently accompanied by signs of thyroid disturbance.

The muscular spasms, therefore, that precede the onset of rheumatoid arthritis in this group of cases may possibly be due to toxic absorption from the alimentary tract; or, on the other hand, they may be the outcome of excessive thyroid reaction, the gland being stimulated by the toxic products to hyper-secretion.

Rheumatoid arthritis sometimes arises at the menopause; and, in cases of some standing, exacerbations of the disease may occur at this epoch. It is, however, not so common at this critical period as osteo-arthritis, more especially that localized form which attacks the knees.

That mental shocks, continuous anxiety, and worry may determine the onset, or provoke an exacerbation of rheumatoid arthritis, is, I think, beyond question. Nearly all writers from Charcot onwards have remarked the frequency with which some such depressing influence has been at work. In one of my own series, the patient, a woman, developed rheumatoid arthritis within a few days of seeing her husband killed by an accident; in another, the disease supervened rapidly in a female who, whilst skating, fell through the ice, and received an acute fright as well as a severe chill.

In several instances conjugal infelicity has appeared to be the starting-point of the disease, several cases in my own experience having been attributed by the patients to grief resulting from enforced separation.

Again, as Garrod observes, the nursing of sick friends or relations for prolonged periods frequently appears responsible:

and often, indeed, no other etiological factor can be ascertained.

As to the exact part played by such factors in the production of rheumatoid arthritis we know little or nothing. But I would suggest that these depressing conditions operate through their disturbing influence on the secretory mechanisms of the alimentary tract. The experiments of Pawlow and his pupils have demonstrated that for the proper secretion of hydrochloric acid in the gastric juice unimpaired nervous control is essential. Moreover, it has been established that in human subjects subsequently to worry, excitement, or great fatigue, the secretion of hydrochloric acid is reduced.

The loss of appetite induced by depressing emotions acts in the same way by diminishing the amount of hydrochloric acid secreted; and, according to Herter and others, it is probable that in states of extreme nervous exhaustion the secretion of pancreatic juice may be impaired, likewise that of bile. As before shown, these diminished secretory activities occasion enfeebled digestive capacity, promote catarrhal states, and ultimately modifications in the intestinal flora, with their malign consequences of abnormal putrefaction. In the condition of chronic toxæmia thus induced, we find, I believe, the explanation of the etiological relationship of these psychic factors to rheumatoid arthritis.

French authors, Charcot,[21] and more recently Teissier,[22] attach great importance to cold and exposure to damp, and doubtless they add their quota, but these environmental drawbacks were not prominent in my series. In one, the disease was ascribed to sleeping in a damp bed, while in another it was attributed to a severe drenching while riding on a tramcar. I note also in McCrae's series that only in two instances was the affection referred to working in damp surroundings.

The history of a hereditary arthritic tendency was only elicited in a few instances. McCrae, however, in his bulletin, states that there was a definite history of arthritis in forty-four cases, and he came to the conclusion that in a slightly larger percentage of such cases the disease started at an earlier period in life than in those in which no such hereditary predisposition to arthritic affections were present.

Tubby states that he has frequently been able to confirm the truth of Lane's contention, that a mixed inheritance of gout and tubercle is often forthcoming in sufferers from rheumatoid arthritis. For myself I am inclined to agree with those who hold that a neuropathic ancestry is more distinctive of these cases.

From the foregoing remarks it will be seen that the etiological problem presented is an extremely difficult one, solution of which is impossible in the present state of our knowledge. While leaning towards the toxæmic theory of its origin, one cannot but be sensible that our knowledge of the more intimate chemical changes involved is very vague, and forbids any pretension on our part to an exact etiology of the affection.

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CHAPTER IV.

BACTERIOLOGY.

It is to Max Schüller[1] that we are indebted for the suggestion of a microbial origin for certain cases of rheumatoid arthritis. His first contribution on the subject was issued in 1892, and communicated to the XXI. Congress der Deutschen Gesellschaft für Chirurgie. He then drew attention to the fact that he had in a certain group of cases noted the presence constantly of organisms in cut sections from the tissues of the joints.

Anatomically these joints were characterized by a condition of villous hypertrophy and generalized thickening of the synovial membrane. Histological section showed in addition that the endothelial cells had undergone proliferation, aggregating themselves into groups enclosing numerous small vessels, which differed, however, from those met with in tuberculous tissues.

The micro-organisms were detected both in and between the cells of the newly-grown hyperplastic tissues of the synovial membrane, and also of the villous growths.

It now remained for him to identify the organisms seen in cut sections with those cultivated by him from the joint fluids, and in a later contribution, in 1893,[2] he announced that this further step had been accomplished.

Adopting strict antiseptic precautions, his mode of procedure was to allow the fluid obtained by puncture from the joint to drop through a cannula directly on to media in tubes and plates, and by this method he obtained organisms identical with those noted previously in histological sections.

He describes the bacilli as "short plump rods showing polar granulation," this latter condition, however, not denoting spore formation, which apparently only took place in old cultures.

As a rule the organisms presented a dumb-bell shape, but in exceptional cases the rods were very short and clumsy, exhibiting but slight mesial constriction, and resembled gonococci, while in others they showed a likeness to diplococci. They might lie singly or in pairs like diplobacilli. The rods measured 1—2 μ by ·25—·75 μ, staining readily with carbol-fuchsin, methylene blue, or Bismarck brown, but not so easily with methyl violet. In these, his earlier publications, Schüller made no reference to Gram's method, but in 1906[3] he states that the organisms are stained by this reagent, and that he uniformly employed it. Whether cultivated in gelatin stabs or on agar or potato, a white granular formation ensued, and the cultures, even after the lapse of ten or twelve months, retained their infective potency.

When introduced into the joints of rabbits, lameness followed, with enlargement of the joints, which proved to be permanent. After the lapse of two months the animals were killed, but prior to death the original organisms were obtained from the joints experimented upon. At no time were the injections followed by pus-formation, but the synovial membrane showed villous growths in places, and in sections from these the organisms were found.

Joints which had not been inoculated showed no change from the normal, and joints into which other organisms had been similarly inoculated did not manifest the same morbid appearances.

With regard to the pleomorphism displayed by the organism, Schüller remarks that inasmuch as the different forms were to be found not only in the tissues of the same case but also frequently in different cultures, he felt justified in concluding that the various types were but modifications of the one organism, and that the varied appearances might possibly represent different stages in the growth of the same organism, or might be due to differences in the media employed for cultivation.

In 1897[4] Schüller contributed a paper dealing with the bacteriological findings in sixteen arthrotomies performed upon the subjects of "chronic villous arthritis." In all of these the bacillus was demonstrated firstly by smears, and later on in sections from the villous excrescences.

He gives particulars of a method by which the organisms can be readily demonstrated in the synovial fringes when exposed during operation. The fringes, when protruding from the wound, are washed with sterile water or $HgCl_2$, incised with antiseptic precautions, and then a sterile glass slide is drawn across the freshly cut surface. The smear is air-dried, and carbol-fuchsin is employed for staining.

The author emphasizes the fact that the organisms are to be found much more frequently imbedded in the tissues than in the joint fluids, and consequently, in aspirating, the needle should not be thrust into the joint cavity, but only into the synovial tissues.

In a series of twenty-three aspirations performed by him, negative results were obtained in six only. He points out that in some rare instances gonococci or pyogenic cocci may be present as well as dumb-bell bacilli, but in such cases he noted that the former organisms lie at a deeper level in the joint tissues.

He rejects the idea of these pyogenic organisms having any causal relationship to the disease, and supports his contention by the fact that pyogenic cocci are often abundant in joints affected by tuberculosis, while the tubercle bacilli themselves may be very scanty.

These examples of secondary infection were especially to be met with in those cases in which capsular atrophy and patchy villous formation were found, as opposed to the other type in which there were chronic capsular thickening and synovial hyperplasia.

In 1906 appeared the *résumé* of his work up to that date, in which he states that his bacillus has been found in 150 out of 230 examples of chronic villous arthritis.

In 1893, Dor[5] cultivated *Staphylococcus pyogenes albus* and a gelatin-liquefying organism from the shoulder-joint of a man suffering from " arthritis deformans " with villous hypertrophy, and suggested that certain forms of the malady are of infectious origin.

Bouchard and Charrin (1891–4)[6] noted the presence of pyogenic cocci in twenty-two cases of chronic joint disease.

In 1896, Bannatyne and Wohlmann[7] discovered a bacillus which they considered to be the cause of the disease. Their observations were confirmed by Blaxall,[8] who succeeded in cultivating the organisms, and arrived at the following conclusions :—

1. In the synovial fluid in eighteen cases of rheumatoid arthritis an organism has been discovered which is constant in its characteristics.

2. The organism is a minute bacillus exhibiting marked polar staining. It is difficult to stain, and is easily decolorized.

3. The organism can be grown in culture media and presents striking characteristics. In beef broth it gives the appearance of gold dust, and on agar-agar and serum its growth is almost invisible. It does not grow on nutrient gelatin at ordinary temperatures.

4. It is present in the blood in severe cases.

5. It has not been found in the synovial fluid from distended joints due to other causes.

The average length of the organism is 2μ and the average breadth $\cdot 6\mu$. It is non-motile and does not occur in chains. It is completely decolorized by Gram's method, but is stained by carbol-fuchsin or methylene blue. Mice, guinea-pigs, and rabbits were inoculated, but without definite results. In 1896[7] Bannatyne stated that he had found the same bacillus in sections from the cartilage, synovial membrane, and periarticular tissues.

In the same year Chauffard and Ramond[9] recorded seven cases of " arthritis deformans " with glandular enlargement, stating that in scrapings from the enlarged glands and in films prepared from the fluid of affected joints a short thin diplobacillus was found. It stained readily by ordinary methods, and was not decolorized by Gram's method.

Inoculations of triturated portions of the glands into culture media and animals proved negative. The enlarged epitrochlear glands were examined microscopically, and fibroses were found, but no infective nodules were noted and no well-marked degeneration.

The search for organisms was only pursued in one of the cases; consequently, as Fayerweather very pertinently observes, the report of this group of cases possesses but little bacteriological value.

Von Dungern and Schneider,[10] in 1898, recorded a case of chronic arthritis deformans from Bäumler's clinic. The clinical history of the case was briefly as follows :—

The patient, a male æt. 45 years, suffered when twenty-eight years of age from an acute polyarthritis with an endocardial lesion, the illness lasting for five months. No recurrence took place for twelve years, when a fresh attack supervened, which resulted in permanent articular changes involving those of the spinal column. Five years later the patient died, and at the autopsy the synovial membrane showed typical changes, with atrophy of the cartilages. The bacteriological evidence in this case is based upon a study of the fluid obtained some months before death from the finger joints, and also from blood withdrawn from one of the veins in the arm.

This procedure was supplemented by an examination (eighteen hours after death) of fluid from several joints, heart blood, spleen, liver, kidneys, and mucus from gall-bladder. A small diplococcus was demonstrated in smears from the contents of the gall-bladder, and also in the joint fluids and liver, and besides these diplococci there were obtained from the liver and gall-bladder another organism resembling in many of its characteristics *Bacillus coli.* From the fluid aspirated during life from the joints, a non-pathogenic coccus was isolated, but in that obtained post mortem there was found not only the diplococcus, but also a non-pathogenic bacillus.

In 1900, Fernet and Lacapere[11] discovered pneumococci in the joint fluid of a woman suffering from pneumonia who afterwards developed rheumatoid arthritis.

In 1901, Painter,[12] dealing with pathological lesions in rheumatoid arthritis, states that both he and another observer had failed to find Bannatyne's bacillus in cases of this malady.

In 1902, Poynton and Paine[13] described a diplococcus found by them in a case of rheumatoid arthritis, and by intravenous inoculation of rabbits produced not only a severe multiple arthritis, but also an osteo-arthritis of the monarticular type. In another case of arthritis of the knee the same observers, in conjunction with Page, isolated diplococci indistinguishable from that of rheumatic fever in the exudation from the knee joints, and cultures were made. Both rheumatic and rheumatoid changes were produced by the micro-organisms injected intravenously into rabbits.

In the same year Hale White[14] obtained a coccus from the left knee joint of a case of acute rheumatoid arthritis. The organism was non-pathogenic, it was apparently not similar to that described by Poynton and Paine, nor did it resemble the more usual cocci. It grew chiefly in pairs, not in chains, and was stained by Gram's method.

From the mesenteric gland in this case a small non-motile bacillus, non-pathogenic to mice and rabbits, was isolated.

A streptococcus was found by Gask in a case of rheumatoid arthritis in 1903.

Crawford and Malim, working at the Royal Mineral Water Hospital, Bath, aspirated the joint fluid in forty-eight cases. They embodied their results in a paper read before the Bath and Bristol branch of the British Medical Association, but no evidence of a specific organism was forthcoming; the majority of the cases proved sterile, and in the few cases from which organisms were cultivated it was thought that they were the results of accidental contamination. The strictest aseptic precautions were taken, and standardized media were used. This piece of work has, unfortunately, not been published by the authors.

In 1904 a report of a series of 110 cases of arthritis deformans from Osler's clinic was issued by Dr. McCrae, and in spite of the greatest care in following the methods given by Blaxall this observer never succeeded in obtaining any results from cultures.

In 1905 an exhaustive *résumé* of the bacteriological work done on this subject was brought forward by Fayerweather,[15] to which I am indebted for much information. Besides critically analyzing the work of previous observers he contributes fresh evidence in support of the microbic theory.

Taking a series of nine examples of the various clinical types of arthritis, he endeavoured to grow cultures from the joints on blood serum. In four of these he obtained positive results; the remainder proved negative. Of the former, one was a case of acute articular rheumatism, the remaining three were examples of Schüller's "polyarthritis chronica villosa." Leaving aside the acute case, in the remaining three he obtained three distinct organisms, which were isolated in pure culture from the synovial fluid. In two cases their presence was further demonstrated in the synovial tissues, and all three were cultivated on artificial media.

In two of these cases, the organism, after introduction into the joints of rabbits, initiated acute and chronic processes clinically and pathologically identical with those induced in the human joint. Pure cultures of the same organisms were isolated repeatedly from the animal joints, and sections from the synovial tissues showed the different organisms *in situ.* In another case, though recovered in pure culture from the animal joint, its presence had failed to provoke any marked change in the joint tissues. In all three, large doses intravenously inoculated proved ineffectual.

It is noteworthy also that in two of the above cases the villous character of the arthritis was established at operation, Dr. Painter, of Boston, having in both cases had recourse to surgical intervention, in the course of which the synovial cavities of the knee were laid open and the villous growths thereby exposed to view.

Passing to a consideration of the group, comprising five, in which attempts at culture had failed, it is interesting to note that two of them were examples of atrophic arthritis (Goldthwait), the remaining ones being respectively cases of acute articular rheumatism, infectious arthritis following influenza, and one of chronic infectious arthritis.

It is only fair to add that in this last case an organism presenting considerable resemblance to those isolated by Schüller and Blaxall was isolated, but the author refrains from including it in the group giving positive results, as there appeared to be some doubts as to the complete exclusion of contamination.

In reviewing the results obtained by these various workers it is all-important to ascertain as far as possible the salient clinical features of the cases upon which they conducted their bacteriological researches, and more especially of those in which positive evidence of the microbic origin was forthcoming.

Dealing first with Schüller's group, this author makes it abundantly evident that he considered them to form a clinical entity quite distinct from arthritis deformans, which term he confines to those cases showing osteo-arthritic lesions, basing his claim on the fact that in his group of cases no essential changes occur in the bony or cartilaginous structures of the joints. He emphasizes his contention by applying to them the special term "polyarthritis chronica villosa bacillaris."

He states that the disease begins, as a rule, in the smaller joints of the fingers, that its onset is insidious and never attended by fever. The condition is a gradually progressive one, involving the wrists, elbows, shoulders, ankles, knees, and hips. It is attended by very marked early atrophy of muscles, while the cartilages of the joints, as seen at operation and by Roentgen rays, remain unchanged. Pigmentation is also to be observed in some cases. The hypertrophy of the villi leads to irregular swelling of the joints, and the synovial processes, according to Schüller, are easily palpable.

Passing in turn to review the clinical features of the cases investigated by Bannatyne, it appears that, excluding the stress laid by Schüller upon the high degree of villous hypertrophy present in his group, both he and Bannatyne are at one in postulating an absence in their series of any bony or cartilaginous changes, as the following quotation from the latter shows: "The micro-organism has been found only in the acute polyarticular cases with soft synovial enlargement of the joints, accompanied by muscular atrophy, by cold sweating palms, by pigmentary changes, by marked cachexia, by a rise in temperature, and possibly by some enlargement in the glands. I have not found it in chronic cases with outgrowth of bone and lipping."

In comparing these separate descriptions, one notes that apart from the lack of essential bony and cartilaginous changes common to both types, the general clinical features of the two groups show a striking resemblance to each other, so close indeed that one is, I think, justified in assuming that any difference that does obtain is one of degree rather than kind.

In other words, that while both series are alike made up of examples of rheumatoid arthritis, those of Bannatyne's are of a more acute character than those of Schüller's.

The "villous" overgrowth so emphasized by Schüller obviously cannot be considered as clearly distinguishing his group from Bannatyne's, for, as before stated, all grades of it are comparatively common in rheumatoid arthritis, and for that matter, in other joint affections.

My plea for their identity is strengthened in that Fayerweather, in his clinical account of "polyarthritis chronica villosa," states that occasionally enlargement of the lymphatic glands, a moderate leucocytosis, and a quick pulse are to be met with. If to this be added the same tendency in their later stages to increasing crippledom from distortion of joints and muscular contracture, the identity of Schüller's cases with those of Bannatyne seems established.

Unfortunately for the conclusiveness of the microbic theory so strongly advocated by these observers, the bacteriological findings by both lack adequate confirmation, as do similarly those of all other workers in the same field.

Reviewing the organisms themselves collectively, one is struck by the variety they display, and presuming the disease to be infectious in origin, the only inference to be drawn is that not one only but many different organisms may produce the disease, and indeed there is nothing unreasonable in the assumption, for this we do know, that different micro-organisms may give rise to identical clinical and pathological phenomena, as in pneumonia and endocarditis. In other words, as Kanthack expresses it, "We may have clinical or pathological identity without bacteriological identity."

It is, however, more difficult to account for the flatly contradictory results obtained by different observers, even when working along the same lines of technique, as shown by comparing McCrae's, Painter's, and other observers' fruitless search for organisms, with the positive results that attended Bannatyne's and Blaxall's researches.

Pending, therefore, greater harmony in the results of bacteriological researches, one is driven to the conclusion that the infective theory of the origin of rheumatoid arthritis, though strongly indicated by certain general clinical features, such as the frequent history of previous infections, glandular enlargements, and so forth, is still wanting in those conclusive bacteriological data on which alone such a conception of its nature could be established beyond cavil.

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CHAPTER V.

MORBID ANATOMY.

THE clinical, as opposed to the anatomical, differentiation of the diseases comprised under the term "arthritis deformans" has been tacitly acknowledged since the time of Charcot.[1] That authority, as we know, recognized at least two main varieties—chronic progressive articular rheumatism and chronic partial articular rheumatism; the former corresponding approximately to the condition we call rheumatoid arthritis, while the latter is represented by osteo-arthritis. Notwithstanding his clear definition of their clinical dissimilarity, Charcot held tenaciously that these two clinical entities were but different expressions of one and the same underlying morbid process, and consistently with this view assigned to them a common pathogeny; while their anatomical lesions, he claimed, though varying in degree, were identical in nature.

This conception of the essential pathological unity of the two joint disorders, despite their clinical distinctness, has endured to the present time, being adhered to with few exceptions by most authorities. The consequence of this is that when we review the pathological accounts of the subject, as given by the older authors, it is difficult, though not impossible, to extract from them a morbid anatomy of rheumatoid arthritis as distinguished from one of osteo-arthritis. While the anatomical lesions of the latter are well ascertained, unfortunately our knowledge of the morbid anatomy of the former, at any rate in its early or more active phases, is extremely meagre. This is, doubtless, largely due to the fact that, owing to the rarity of a fatal termination in this period of the disease, those changes which have been recorded as pathognomonic of rheumatoid arthritis are in reality those pertaining to the more advanced stages or, as Dr. Garrod[2] terms them, the stage of sequels. At this stage the majority of the anatomical lesions produced are similar to those which might have resulted from any long-continued degenerative joint process ; and the extensive destructive changes in the articular structures, being attended in some instances by new bony proliferation, gave rise to a certain rough similarity to osteo-arthritis, which led to their being erroneously regarded as more or less identical with that condition.

Recent research, however, tends more and more to indicate that their clinical distinctness is probably the outcome of equally distinct morbid processes.

In entering upon a brief retrospect of the earlier contributions to our knowledge of the morbid anatomy, it is interesting to note that, as far back as 1798, Gotz[3] recorded the autopsy of a case which presented the anatomical lesions we now recognize to be distinctive of rheumatoid arthritis as opposed to osteo-arthritis. The condition was one of chronic polyarthritis with spinal involvement. In addition, marked subluxations and malpositions of the limbs, with symmetrical ankylosis of the carpal articulations, were observed, while eburnation of the articular ends and osteophytic outgrowths were not noted.

Hoffa,[4] reviewing this case, concluded that it was an example of rheumatoid arthritis, or, as he would designate it, primary chronic progressive polyarthritis.

Similarly, Cruveilhier,[5] in his pathological anatomy, describes a case presenting the same marked distortion of joints and erosion of cartilage, but no bony proliferations.

The fact that Adams[6] felt justified in dividing his examples into two categories, a constitutional and a local variety, carries with it, I think, the legitimate inference that this observer recognized the desirability of distinguishing the local or osteoarthritic from the constitutional or rheumatoid type. Indeed, when defining Case 19 of his series, he refers to it as an example of " chronic rheumatic arthritis of the right knee-joint of the hypertrophic form."

From Cornil and Ranvier's[7] reflection that in the variety of chronic rheumatism they investigated bony ankylosis of the articular ends did not occur, it may be fairly concluded that they were dealing in the main with osteo-arthritis ; and the correctness of this supposition is, I think, heightened by consideration of their well-known description of the histological changes in the bony and cartilaginous structures.

According to Hoffa, it is to von Volkmann[8] that we are indebted for the first clear differentiation of the anatomical lesions of rheumatoid arthritis from those of osteo-arthritis.

This authority first established the fact that the initial changes in the former began in the synovial membrane, the cartilages being only affected secondarily. He noted the primary thickening of the periarticular and synovial tissues, and the subsequent atrophy and induration of the same, likewise the occurrence of fibrous and bony ankylosis.

Moreover, in his work he contrasts the features of this group of cases with those distinctive of osteo-arthritis, of which he gives a detailed description.

Hueter,[9] also, from his anatomical researches, confirmed Volkmann in his main deductions.

To Samaran's[10] work, however, something more than passing notice is due; for, as Hoffa states, it is of the greatest value. This author gives the post-mortem accounts of two cases, which I take the liberty of transcribing from his exhaustive monograph.

Case 1.—Patient, 73 years old, inmate of a hospital for incurables, and the subject of polyarthritis. Great emaciation, with atrophy of organs, was present. The hands were markedly crippled, with pronounced disease of all the joints, and marked stiffness. The bones were brittle, rarefied, and very rich in fat. In the right shoulder and both elbow-joints a soft granulation-like tissue was present, while the cartilage in the same was only recognizable in a few spots. In the ankylosed joints the cartilage was replaced by a tissue, partly fibrous, partly of a reddish gelatinous consistency, uniting the ends of the bones. There was no enlargement of the articular ends of the bones, which, on the contrary, appeared rather diminished. Atrophy of the bones was general, and of such a high grade that small portions of the same floated in water.

Microscopic Examination.—The cartilage was transformed into mucous and connective tissue. This was apparently brought about by a solution of the cement-substance of the cartilage fibres. In the formation of mucous tissue the cartilaginous fibres became dissolved. Extensive new formation of blood vessels was observed.

Case 2.—Woman, 44 years old. The morbid changes noted were similar to those seen in the former case. The synovial membrane was greatly vascularized and changed into a soft tissue. The cartilage was replaced for the most part by a soft vessel-containing tissue, uniting the two articular surfaces. There was no formation of new bone to be recognized.

Samaran in his deductions maintains that the cartilage in this disease behaves quite differently from that in osteo-arthritis; thus, marginal hypertrophies and localized thickenings are wanting; hence, there is an absence of joint-deformities. Another conspicuous differentiating feature is to be found in the atrophic character of the changes in the bones.

Waldmann[11] also contributes the results of an autopsy on a patient, the subject of the "atrophic form of chronic articular rheumatism."

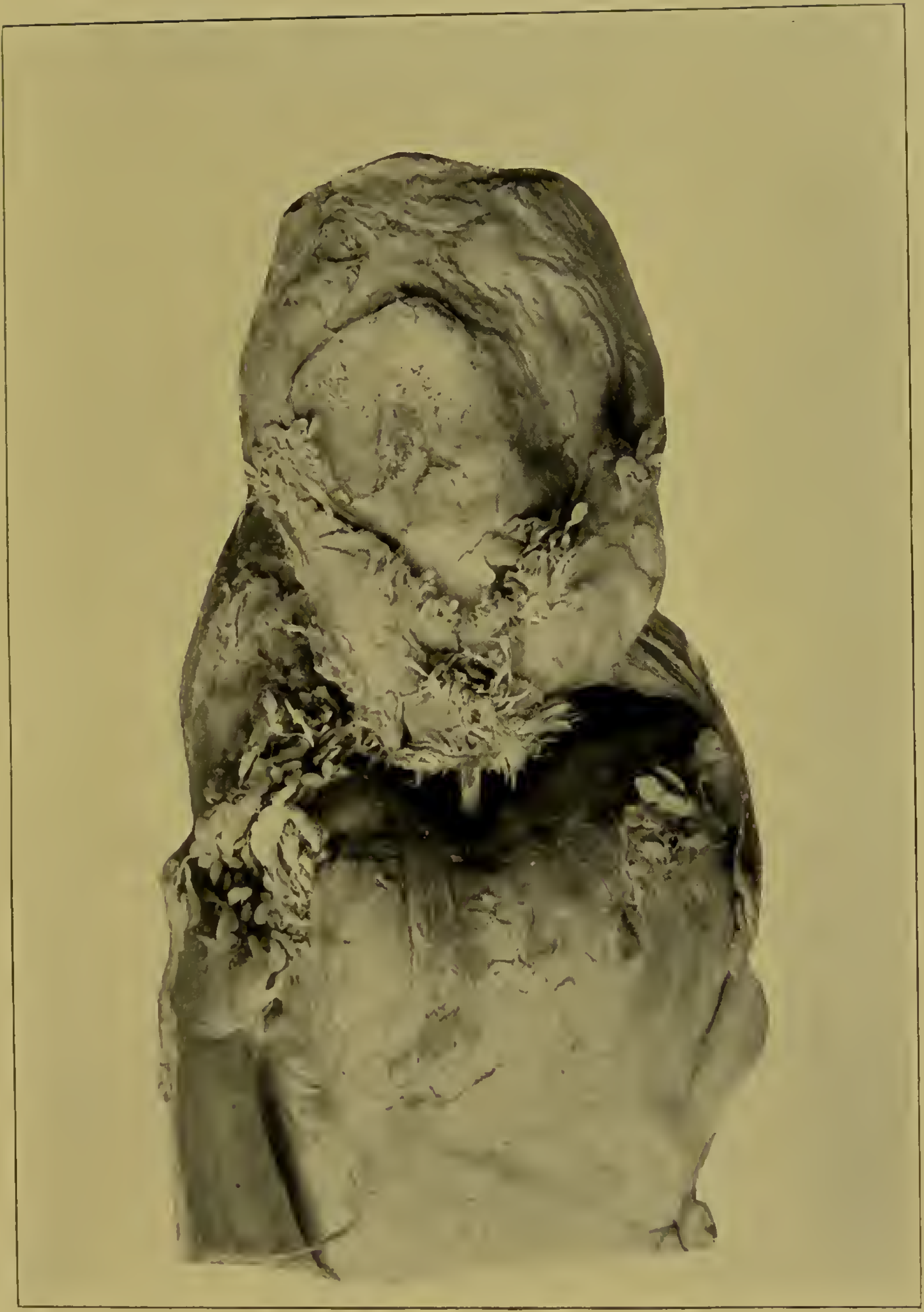
The following changes were observed: Moderate thickening of individual joints; nowhere ankylosis. The joint-capsules were sclerosed, as if felted. Of the synovial membrane and cartilage there was hardly a trace to be seen in the shoulder, elbow, knee, wrist, and finger-joints. In isolated articular extremities—for example, in the metacarpo-phalangeal joint of left index finger and right wrist-joint—there were present in the bony ends somewhat deeply infiltrating and irregular deficiencies, with islets of normal bone. No trace of pus was observed. The shape of the articular ends was on the whole unaltered.

The chief features that call for notice in this case are the regressive character of the osseous and cartilaginous changes throughout, with a consequent absence of deformity of the joints.

Muller,[12] as a result of extensive operative experience in these conditions, arrives at the following conclusions. Deformities, he says, may be completely absent in the early stages of rheumatoid arthritis, even though the disease may have existed for five or six years. The cartilage is, as a rule, either unaltered or only shows simple erosion, or transformation into connective tissue. Only in one case did he note bony outgrowths, and those on the patella; while the condyle of the femur showed slight eburnation.

Kachel[13] also contributes two undoubted examples of rheumatoid arthritis, which he submitted to detailed anatomical investigation. In the first of these, a woman, 57 years of age, notwithstanding the previous existence of the disease for twenty years,

PLATE I.



Knee Joint, showing erosions of the cartilage both on the femur and patella, in some parts exposing the bone. Synovial membrane is beset with elongated synovial tufts of irregular shape, some of them branched. A fibrous band is seen passing across the upper part of the synovial cavity from one wall to the other. No bony outgrowths are present, notwithstanding the existence of the disease for eleven or twelve years.

(From R.C.S. Museum, No. 1902.)

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there was an absence of cartilaginous and bony proliferation in the joints. On the contrary, general simple atrophy of the bony tissues was present.

In the second case, a woman, 52 years old, the disease commenced, when twenty years of age, in the finger-joints of one hand. Subsequently the morbid process became generalized, becoming symmetrically distributed, with marked muscular atrophy.

Here, again, the distinctive changes were those of thickening and villous overgrowth of the synovial membrane, with agglutination of the opposed surfaces of some joints by fibrous tissue. Bony ankylosis was nowhere to be found. Additional features were atrophy of the cartilages and bones, with absence of proliferation of these structures. Kachel ascribed the atrophy to prolonged inactivity; and possibly a portion of it, he thought, might be referred to nutritive disorders (arteriosclerosis).

This brief retrospect of the contributions dealing with the morbid anatomy of the disease shows that the anatomical differentiation of rheumatoid arthritis from osteo-arthritis, far from being of modern date, was well known to the older authors, especially those of the German school. Indeed, when we come to consider the more recent anatomical investigations on the subject, one cannot help feeling that but little fresh has been added to the pioneer work of Volkmann, Samaran, and Waldmann.

Amongst recent observations one of the most important was made by Hale White,[14] who had the opportunity of ascertaining the condition of the joints in a comparatively acute case, the subject, a young woman, having died of an intercurrent malady.

Examination of the joints revealed an overgrowth of fibrous tissue in the periarticular and ligamentary structures, both being much thickened.

These, with the alterations in the synovial membrane, constituted the salient anatomical lesions; the bones and cartilages being only affected to a minor degree.

In one of the proximal phalangeal joints, the synovial membrane was the seat of newly-formed fibrous tissue, and showed evidences of both recent and chronic inflammatory changes.

The membrane covering the cartilage showed thickening and formation of fibrous tissue, also some hypertrophy of the synovial fringes, and in the neighbourhood of, or beneath, these, the cartilage showed signs of thinning and erosion, the underlying bone in some places being exposed. The absorption of the cartilage was probably due to pressure by the thickened villous processes. In addition to these changes in the cartilaginous structures, foci of inflammation were to be met with in the bone, the cancellous tissue of which was more open than usual. Microscopical section showed that these foci were the seat of a small-celled infiltration, while the fat cells were markedly decreased in number. Compared with the inflammatory changes in the synovial membrane, however, those in the bony structures were very slight. In the right knee-joint there was an excess of synovial fluid found, and the cartilage covering the external condyle presented a slight erosion; but there were no outgrowths from the bone or the cartilaginous edge.

According to Bannatyne, the following changes were noted by him in an acute case of rheumatoid disease :—

Much thickening of ligamentary and periarticular tissues, with redness and injection of the synovial membrane, which appeared softened, thickened, and pulpy. The cartilage had undergone softening, and was the seat of an erosion occupying one-third of the articular surface, while the underlying bone was red, eroded, and soft. In the later stages villous hypertrophy takes place.

These excessive villous formations, so extremely common in rheumatoid arthritis, are usually generalized over the synovial membrane: unlike those ensuing after trauma, which are usually more localized in distribution.

This condition has been exhaustively studied by Schüller,[15] and Painter and Erving.[16] The former claims that it is a distinct disease associated with a particular micro-organism; the latter, however, like most observers, consider that it is not a distinct entity, but is a condition more commonly associated with certain chronic diathetic diseases, the two principal of which are rheumatoid arthritis and osteo-arthritis.

Their characteristics are as follows: The hypertrophied fringes vary widely both in their appearance and size. The smaller and more delicate, purple in colour, are attached by a slender pedicle to the synovial membrane, whose surface may be studded with these elongated processes. On the other hand, they may be of considerable size, the so-called lipoma arborescens, when they present a yellowish colour and are coarsely lobulated in shape. These, in addition to their usual pedicle, may be attached by adhesive inflammation over a large area of the synovial membrane. Microscopic examination of the fringes shows that they are enveloped by a layer of endothelial cells, internal to which is a groundwork of connective tissue infiltrated with small round cells, and abundantly supplied with thick-walled blood-vessels. Progressing from the periphery inwards this connective tissue becomes replaced by fat cells aggregated into areas of varying size, with intervening bundles of connective tissue. The vessel-walls show endarteritic changes, their lumina being often obliterated, especially in the more central portions, where fatty deposits are most abundant.

With regard to the synovial effusion, it varies greatly in amount; according to Painter, there is very rarely any excess of fluid in the so-called atrophic, or rheumatoid, arthritis; but the majority of observers are inclined to believe that an excess of fluid is not so very infrequent.

In some cases that came under my own observation excess of fluid was present, and usually of a viscous nature, not infrequently blood-stained and yellowish in colour. Fibrinous flocculi, or coagula, are sometimes to be met with.

Bannatyne[17] states that bacteria in large numbers are to be met with in the fluid. According to Fagge the effusion of blood into a rheumatoid joint was noted by Goodhart. Mansell Moullin[18] and Lunn[19] have recorded instances in which the effusion was of a purulent nature; but such are extremely rare, and in all cases due to secondary infection. McCrae[20] narrates an instance in which the knee-joints of a man, the subject of arthritis deformans for over two years, became the seat of an abundant purulent effusion in sequence to a general pneumococcal infection.

The foregoing unfortunately represent all the pathological observations at our disposal as to the morbid changes incidental to the earlier, or more acute, phases of the disease.

The pathological changes we are now about to describe represent those met with in the more chronic types of rheumatoid arthritis and those characteristic of the terminal stages of the affection.

Hoffa gives in great detail the macro- and microscopic anatomical changes in chronic rheumatoid arthritis, of which we cite the following as an example : Patient was a girl, 29 years of age, with a polyarticular affection which had existed for six years. His colleague, Wollenberg, performed an arthrotomy on the mid-phalangeal joint of the fourth left finger, which articulation had been diseased, according to the patient, for one month, at the outside two months.

During the course of the operation a small subcutaneous nodule was excised, and found to consist of thickened connective tissue. The periarticular tissues were thickened, gelatinous, and greyish-red in appearance. The fibrous layer of the capsule was thickened, likewise the synovial membrane, which presented a gelatinous appearance, and was the seat of villous overgrowth. There was no intra-articular effusion. The interphalangeal cartilages were of a normal bluish-white colour, apparently somewhat rarefied in their central portions. Their surface was not markedly roughened. Proliferative processes were absent, the shape of the articular ends being quite unaltered.

Pieces of the synovial tissue and fibrous capsule were removed for examination ; likewise a piece of the marginal cartilage.

Changes in Synovial Tissue and Fibrous Capsule (*stained with Hæmatoxylineosin*).—Microscopical examination of the synovial membrane showed numerical increase and hypertrophy of the villi, with many new blood-vessels. Extensive and peculiarly dense fibrous overgrowth was present, well supplied in parts by new blood-vessels. Large perivascular cellular infiltrations were noted, these being more abundant in the subintimal cell-layer and the villi than in the fibrous tissue. These cellular aggregations in places gave rise to lymphomalike nodules, in the midst of which one or more blood-vessels were situated.

When present in the villi they gave to them a club-shaped appearance. Evidences of hyaline degeneration were present in some of the larger villi. These perivascular infiltrations were found to consist of densely-packed small mononuclear leucocytes with large deeply-staining nuclei and delicate protoplasm, while here and there polynuclear leucocytes were noted. Entering into the formation of the nodules were also fixed connective tissue cells. Although more marked in the nodules, leucocytic infiltrations were diffusely scattered throughout the tissues. The subintimal cell-layer, as well as the villi, consisted of a loose, finely-reticulated connective tissue, rich in cells. Towards the fibrous layer the connective tissue-cells became more numerous and larger, proliferating to such an extent as to resemble a thick granulation tissue.

The thickened fibrous layer consisted of connective tissue rich in nuclei, long, and spindle-shaped. The ground substance had here and there assumed a somewhat homogeneous character. In the fibrous layer, as in the synovial layer, similar leucocytic infiltration, though more scanty, was observed. Areas of mucous degeneration were found in the fibrous tissue: usually, but not invariably, in the neighbourhood of blood-vessels.

Changes in the Joint Cartilage (*similarly stained*).—The cartilage cells in the deeper layers were of normal size; here and there their capsules had undergone calcification. Towards the superficial layers the cells became more scanty, frequently showing a distinct diminution in nuclear staining. At this level the cells were irregularly arranged.

These changes were most marked near the free margin of the cartilage, while towards the centre a more normal distribution existed. At the free border cartilage-cells were remarkably scarce. In the most superficial layers the margin of the cartilage was composed of a disintegrating crumbling ground substance, in places of a slaty colour, with numerous, for the most part degenerated, cells, which with their nuclei stained badly. The capsules of the cartilage-cells were here usually absent. In this area of the cartilage, which had undergone such degenerative changes, no active proliferation on the part of the cells had taken place.

Painter,[21] as the result of various surgical procedures conducted on joints the seat of atrophic or rheumatoid disease, noted certain facts of which the following constitute the main features:—

The synovial membrane was the seat of more or less extensive villous formations; occasionally, bony and cartilaginous masses were present in their substance, and in one instance the synovial membrane was covered with islands of cartilage, ranging in size from a pea to a thumb-nail. The cartilage here and there showed varying degrees of erosion, the underlying bone in some regions being laid bare.

In the case of a young woman, 26 years of age, with extensive joint disease, in whom excision of the elbow was performed, the cartilage covering the particular ends of the radius and ulna had almost completely disappeared. That the shafts of the bones participate in the rarefactive process was well illustrated in the case of a young man, 26 years of age, the subject of the disease for five or six years. Partial ankylosis of the hip had resulted; and in order to secure motion, and correct the faulty position, excision was performed. The portion of the shaft of the femur removed was found to be spongy, loaded with fat, and its cortical layer so thin that it could be easily crushed between the fingers.

Histological examination of the structures, derived from these and other operations, revealed the following characteristics:—

The hypertrophied villi were the seat of general hyperplasia of the connective tissues, in which occasionally there were areas which had apparently undergone colloid degeneration. The blood-vessels were increased in number, their adventitial coat had undergone marked thickening, and occasionally obliteration of their lumina had resulted. This observer emphasizes the rarity with which leucocytes were to be seen, and that the histological sections when stained for fibrin did not respond.

The ground substance of the cartilage had undergone fibrillation and necrosis in places, and was incapable of being stained, while contiguous portions took the stain readily.

Microscopical examination of the bone showed that the trabeculæ were very much attenuated, while there were very few osteoblasts to be seen.

The bone-marrow cells were apparently normal; but there was a great quantity of fat and some excess of blood-vessels in the marrow.

Recently, Nicholson,[22] of Cambridge, has contributed a study of the morbid histology of some cases of rheumatoid arthritis. After careful consideration of his work it is impossible to avoid the conclusion that the material upon which this observer conducted his interesting researches was of a mixed nature. Thus, under the above title, he describes in detail two groups of cases, one of which, undoubtedly, corresponds to the classical description of osteo-arthritis. This variety, he states, was characterized by eburnation of bone, osteophytic outgrowths, and an absence of bony ankylosis.

The cases constituting his second group should, he suggests, be regarded as a distinct variety of the disease, since fibrous, or even bony, union is the end-result. Moreover, he adds that the shafts of the bone in this class are obviously much wasted, the cortex undergoing marked attenuation, and the cancellous tissue being likewise the seat of rarefactive change.

It cannot be doubted that the cases in this latter category were, strictly speaking, examples of rheumatoid arthritis, inasmuch as they conform in all their anatomical characters to those already known to be distinctive of that affection.

From a review of the foregoing pathological observations by various students of the disease, one is, I think, justified in drawing the following conclusions in regard to rheumatoid arthritis :—

1. That the morbid process begins in the synovial and peri-articular tissues, which undergo thickening, with abundant villous proliferation and new formation of blood-vessels. The cartilage, at any rate in the early stages, remains intact.

2. The changes that ensue in the cartilage and bone are to be regarded as secondary, and are for the most part, as Hoffa says, regressive and passive in character.

3. In some cases proliferative changes take place in the bony and cartilaginous structures, giving rise to irregular bony outgrowths at the level of the articulation. Such, when they occur, are inconsiderable in size, and apparently are in no relation to the duration of the disease.

4. The terminal stage is marked by shrinkage of capsule, with obliteration of the joint-cavity, and ultimately fibrous or even bony union of the articular ends of the bone. The shafts of the bones undergo atrophy, the cortex becoming much attenuated ; while the cancellous tissue undergoes rarefaction, being replaced by scanty thin trabeculæ.

In discussing these conclusions seriatim, one may, in regard to the first, point out that while in rheumatoid arthritis the initial changes are found in the synovial membrane, and the cartilage and bony structures are only affected secondarily, in osteo-arthritis, on the contrary, the primary changes take place in the cartilaginous and bony structures, the synovial tissues being only implicated secondarily.

With regard to the second, while the changes in the cartilage and bone in rheumatoid arthritis are retrograde and passive in character, those of osteo-arthritis are active and proliferative. In short, atrophy of these structures in the former, hypertrophy in the latter, constitutes the distinguishing features.

As to the minor degrees of proliferation that take place in rheumatoid arthritis, it has been suggested that such are to be regarded as evidence of the transition of this condition into one of osteo-arthritis. It is, however, open to question whether these bony proliferations do not admit of another explanation.

The morbid process in the articular ends in rheumatoid arthritis is one of rarefaction and softening.

Consequently, it follows that the involved structures, by reason of this change, are peculiarly susceptible to stresses and strains brought to bear upon them; in other words, the ends of the bones tend to undergo plastic adaptations in their shape as a result of the retraction of the skin and peri-articular tissues, aided by the pull of the related muscles.

From a consideration of X-ray photographs I am inclined to think that this affords a reasonable hypothesis as to the factors responsible for the altered shape in the ends of the bones.

As Hoffa states, the bony proliferations, when they do occur in rheumatoid arthritis, are in proportion to the amount of atrophy that has occurred; and he would regard them as being reparative in character.

Again, were the proliferative changes in rheumatoid arthritis of a similar nature to those in osteo-arthritis, one would expect that they would be in some relationship to the duration of the joint disorder. On the contrary, from observation of the cases recorded in the literature, it would appear that in some instances, notwithstanding the existence of the disease for nearly twenty years, bony outgrowths were absent; whereas, in others of much briefer duration, slight bony proliferations have been noticed.

Touching the question of ankylosis in this affection, one may note that Painter, when dealing with this point in atrophic arthritis, comments on the absence of fibrous adhesions in the early stages. He is of opinion that such are confined to the later stages when contractures have developed, and the joints have been condemned to immobility for prolonged periods.

When, however, as the result of denudation of the articular ends, cancellous tissue comes into contact with cancellous tissue, true bony ankylosis occurs.

To sum up, it may be stated that bony ankylosis occurs not infrequently in rheumatoid arthritis; rarely, if ever, in osteo-arthritis. The immobility in the latter, when present, is due to the inter-locking of osteophytic outgrowths.

From X-ray and anatomical observations it is obvious that there is a tendency in rheumatoid arthritis to obliteration of the joint-cavity from adhesion of the articular surfaces; and Hoffa regards this effacement of the joint cavity as another distinguishing feature from osteo-arthritis, in which the inter-articular space, though diminished, is never wholly lost.

As to the nature of the histological changes in the synovial and other articular tissues in rheumatoid disease, if we except some of the older writers, such as Colles, Todd, and Fuller, who ascribed the disease to some disorder of nutrition, most modern writers have regarded them as of inflammatory character.

In opposition to these, Painter, when discussing the interpretation of the pathological changes observed in his own series of cases, finds it difficult, if not impossible, to reconcile them with those characteristic of frankly inflammatory affections of the same structures.

The chief obstacle, to his mind, consists in an almost complete absence of histological elements in the joint-lesions at all comparable to those pathognomonic of inflammatory processes as generally understood.

Thus, he contrasts the microscopic changes in the bones as seen in one of his cases with those met with in chronic osteo-myelitis, drawing attention to the following points of dissimilarity:—

In chronic osteomyelitis the intertrabecular structure is very much more dense than in rheumatoid arthritis, consisting of hard, fibrous tissue; whereas in rheumatoid disease it is largely made up of fat, with very little fibrous tissue.

The trabeculæ undergo very marked attenuation in rheumatoid arthritis; while they are very little thinned, if at all, in osteomyelitis.

Osteoblasts are extremely numerous in the neighbourhood of healthy growing bone in osteomyelitis; on the other hand, they are extremely rare in rheumatoid arthritis.

In osteomyelitis the cellular elements are made up of round cells, epithelioid cells, and a few leucocytes, the lacunæ being filled up with nuclei of bone corpuscles; while in rheumatoid arthritis there are rarely any such nuclei, leucocytes are conspicuously rare, and epithelioid cells are absent.

In both diseases blood-vessels are very numerous; but in rheumatoid arthritis endarteritic changes are more prominent.

In continuation, he draws attention to the fact that all inflammatory processes with which we are familiar are associated invariably with reparative processes, but he maintains there is no histological evidence of an attempt at repair in the case of the osseous lesions of atrophic arthritis.

He is inclined to think that the histological lesions of rheumatoid disease resemble those of neurotrophic origin. In this supposition many would agree with him, for, as he maintains, the clinical evidence is overwhelmingly in favour of this mode of genesis. This concludes our consideration of the morbid anatomy of the articular lesions.

We now pass on to describe the changes noted in other structures, more particularly those found in the *nervous system*.

In a few instances Folli[23] noted the presence of atrophic changes in the cells of the anterior cornua; likewise Klippel[24], quoted by Massolongo.

Bannatyne also, in one instance, noted degeneration with vacuolation of the ganglion-cells in the same region. Pitrès and Vaillard,[25] in a few cases, found evidence of neuritis in the peripheral fibres, but did not consider that they were causally related to the joint lesions, being probably of the nature of a secondary neuritis.

The most elaborate account of the changes that may occur in the central nervous system is one furnished by Dr. Mott and Mr. Tredgold,[26] and is included by them in an article, entitled, "Some Observations on Primary Degenerations of the Motor Tract."

Because of the exceptional interest of the case, I preface it with a short clinical account.

The patient was a widow, aged 53 years, a charwoman by occupation, who, becoming insane, was admitted into Colney Hatch Asylum.

Her personal history disclosed the following facts: Her joint-troubles began four years previously with pains in the feet, and then in the knees, which, subsequently becoming contracted, prevented her walking. Eighteen months afterwards, the hands likewise became the seat of pains, and marked rigidity of the elbow and hand eventually supervened.

She presented the following features when examined: Movements of the head were very limited, the chin resting on the sternum as she lay in bed. Both hips and knees were flexed and the ankles extended. The internal condyle of the femur was prominent, the end of the bone projecting in front of the tibia, while the patella rested on the outer condyle. The upper extremity of the fibula projected externally. Considerable osteophytic outgrowths were present. Knee and ankle joints were apparently fixed. The big toe was displaced outwards. The four digits at the metatarso-phalangeal joints were over-extended; the proximal phalangeal joints were considerably thickened and fixed. Muscular atrophy of pronounced degree was present in the limbs. The hands were the seat of marked deformity; the terminal phalanx was flexed on to the second at an obtuse angle, the second was extended on the first phalanx, while the first phalanx was flexed on the metacarpal bone; the carpus and metacarpus were flexed to a less marked degree on the bones of the forearm; the hand showed ulnar deviation. The shoulder-joint was rigid, the arm closely adducted, the elbow flexed, and the forearm pronated. Great nodular deformity about the metacarpophalangeal joints and the wrists. All muscles were much wasted.

The skin over the hands had lost its wrinkles; was smooth, red, and cold; but there was no loss of sensation. The finger-nails were small and atrophied. Muscles showed varying degrees of wasting in the following order: Forearm, arm, foot, leg, and thigh. They exhibited no fibrillary twitching. They responded readily to faradism, while K.C.C. was less than A.C.C.

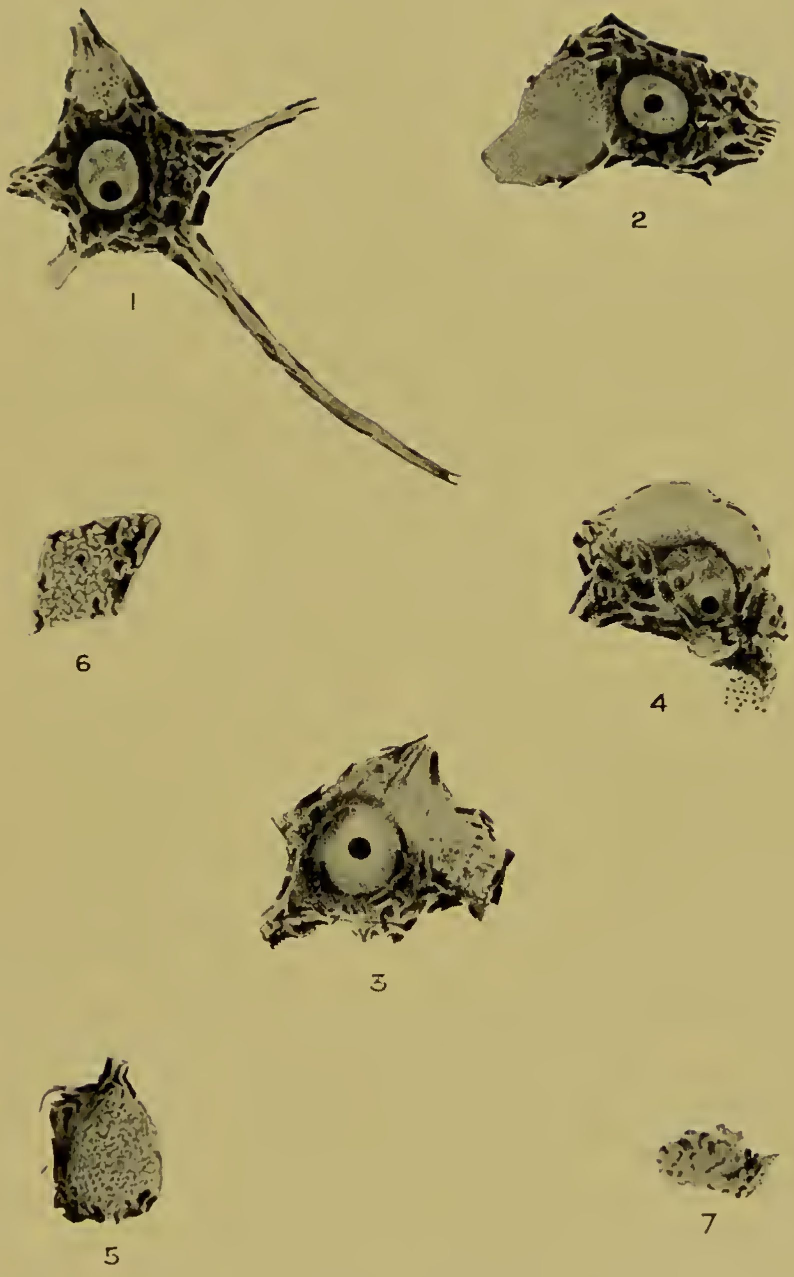
Three months later the patient died of bronchopneumonia. At the necropsy the following changes were noted :—

Pneumonia and recent pleurisy of the right upper lobe. Heart small, but showed no valvular lesions. Great enlargement of liver, with nutmeg appearance on section. Kidneys, specially their cortex, were very pale and obviously fatty. The brain and spinal cord were removed for microscopical examination, and sections stained with Nissl's method showed no change in the cortical cells, nor in the rest of the motor tract down as far as the medulla.

Sections of the spinal cord were examined at the level of the sixth, seventh, and eighth cervical ; the first, tenth, and twelfth dorsal ; second and fifth lumbar ; and first sacral segments. In the grey matter of the cervical enlargement, the large cells of the anterior cornua were fewer in number than normal ; a large number of them showed chronic degenerative change, with large pigmentary deposits. The lumbo-sacral portion of the cord showed exactly similar changes ; many cells showing various degrees of degeneration, being scattered throughout the grey matter (*Plate II*). On the other hand, in the dorsal segments the degenerative processes were much less marked, only a few cells showing any changes. In the cervical and lumbar enlargement there was present very considerable decrease in the fibres forming the network of the anterior horns, especially in the central portion. This change was not present in the dorsal segment. The white matter of all the segments examined showed a slight general increase of the interstitial tissue, especially marked in the neighbourhood of the fissures and vessels ; the latter being in a condition of arterio-capillary fibrosis.

No diminution in the number of nerve fibres, and no sign of any tract degeneration. Membranes showed no change. Anterior roots at cervical and lumbar levels much atrophied. Spinal ganglia from the lumbo-sacral enlargement, when examined and stained by Nissl's method, showed the cells to be somewhat shrunken, shrivelled, and pigmented. Slight increase of fibrous tissue was present throughout the ganglia, but no very definite signs of degeneration, nothing of an acute

PLATE II.



The numbers 1 to 7 illustrate the successive changes in the nerve-cells, stained by Nissl's method.

The cells undergo reduction in size; the Nissl bodies being replaced by yellow pigment staining black with Marchi's fluid; the nucleus shrinks, becomes indistinct, and eventually disappears; the processes get thinner and break off, and ultimately only a small mass of granular pigment remains.

(By kind permission of Dr. Mott).

nature. Both great sciatic and ulnar nerves were examined in longitudinal and transverse section. In the Marchi preparations no recent degeneration was noted, but a considerable diminution of the fibres. Cross section showed that there were quite large areas almost devoid of nerve fibres, these being replaced by connective tissue over-growth.

Muscle.—A section of the thumb muscles stained by Marchi's fluid showed that while a few fibres appeared to be quite healthy, a still larger number were cloudy-looking, their striæ indistinct, though showing no fatty change. In some, however, fatty degeneration was present in typical form. The authors came to the conclusion that the microscopical appearances in this case almost exactly agreed with those observed in two cases, considered in the same paper, of amyotrophic lateral sclerosis, and one of progressive muscular atrophy. The same thickening and fibrosis of the vessel wall was present, while the cells and fibres were the seat of the same chronic atrophy and pigmentary deposits. The one point of difference consisted in this, that in the other three cases the mesial anterior group of cells in the anterior horns was comparatively little affected, the chief change being in the lateral groups ; whereas, in the rheumatoid case, the change was more generalized, there being as many atrophied cells in one group as the other. As to the exact relationship of the anterior cornual changes to the joint lesions, whether primary or secondary, the authors felt themselves unable to give any opinion, but simply record the fact.*

Osler, in his section on "Arthritis Deformans," refers to the following case reported by Triboulet and Thomas[27] from Dejerine's clinic. The patient was a woman, aged 19, who in March, 1894, had a miscarriage, followed by septic infection. Subsequently to this she developed a polyarticular infection, accompanied by the most pronounced muscular atrophy and general cachexia. The patient succumbed to the disease within twenty months of its onset. Systematic examination of the cord disclosed the following changes: Extensive thickening of the pia-mater and arachnoid by connective tissue overgrowth, unattended by any notable alterations in the vessels, except that at certain points a periphlebitis seemed present. These changes, especially marked in the lumbar region, were also present in the dorsal and cervical regions of the cord, especially at the point of entry of the posterior roots and over the posterior half of the cord. The cord itself was the seat of degeneration of the posterior columns, extending from the lumbar region upwards to the cervical region. Most extensive in the lumbar portion of the cord, it involved in the dorsal region only the median portion of the posterior columns, and in the cervical region the tracts of Goll. Many peripheral nerves were examined without revealing any change in the axis cylinders or myelin-sheaths. Lastly, the muscles, notwithstanding the grave degree of atrophy, on histological examination, showed no departures from the normal. Their striation was perfect, the connective tissue showed no modification, in short there was no trace of degeneration, which is somewhat surprising, as the observers remark. They arrived at the conclusion that the primary lesion had its site in the meninges, to which the cord changes were secondary.

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* In a letter to myself Dr. F. W. Mott expresses his belief that the changes in the anterior horn cells of the spinal cord in this case were occasioned in the following manner :—

"The rheumatoid arthritis caused fixation or deformity of the joints, secondary disuse atrophy of the muscles and of the deep nerves sensory and motor, *followed by atrophic changes* in the anterior horn cells."

In his opinion the anterior cornual lesions were not causally related to the joint changes.

In the following cases contributed by Pitrès and Carrière[28] to the "Archives Cliniques de Bordeaux," marked changes were found in the peripheral nerves.

Case 1.—The clinical account runs as follows :—

Patient, a woman, was 40 years of age. Her family history was negative; likewise her personal history, except in so far as in the course of her work as a domestic she had been exposed to considerable cold and damp. The disease began in her left foot, and gradually all the joints with the exception of the hips were involved, even the temporomaxillary joint being implicated. Sensory disturbances were absent, except slight hyperæsthesia over the affected articulations. There were no vasomotor disturbances. The deep reflexes, as far as could be ascertained in joints not ankylosed, were normal. The skin and nails were the seat of trophic changes, and there was present extensive muscular atrophy. Physical signs of tuberculosis were present at both apices of the lungs.

The autopsy disclosed the presence of advanced tuberculous disease of the lungs. The affected joints showed the following changes: Thickening of the connective tissues, some increase in the size of the ends of the bones, eburnation, destruction of the cartilages in places, development of vegetations on the synovial membrane. Portions of nerves from different regions were hardened in 1 per cent osmic acid solution and double-stained with borax-carmine. The cord was hardened in Muller's fluid. The anterior and posterior nerve-roots were normal. The large nerves of the arm showed an increase of badly-staining fibres, with segmentation of the myelin in places, and nuclear proliferation more marked towards the periphery. These changes were most intense in the filaments supplying the fingers. The sciatic nerves on both sides were normal; but their branches supplying the knee-joints were the seat of changes similar to those observed in the nerves supplying the fingers. The lesions were those of parenchymatous and interstitial neuritis. The cord, however, proved to be normal.

Case 2.—The clinical account runs as follows :—

The patient was a woman, aged 57 years. Her personal history was negative, but her family history showed that her mother and grandmother were rheumatic. The disease began in the small joints of the fingers. All the interphalangeal and metacarpophalangeal joints were affected, the hands being much deformed, and maintained in a position of flexion. There was great atrophy of the small muscles of the hands, especially the interossei. The carpocarpal and radiocarpal articulations were intact. The knee joints could only be flexed with difficulty, the ends of the bone entering into them being much enlarged and deformed, the neighbouring muscles atrophied. The other joints were normal. The skin about the affected articulations showed trophic changes, as did also the finger nails. The patient complained of severe pains, and sensations of burning and cold about the affected joints; but presented no objective sensory symptoms, other than slight hypæsthesia on the inner side of the knee joint. She developed a serous pleurisy; was tapped four times; but gradually ran down, and died six months after coming under observation.

Autopsy showed slight fatty degeneration of the heart and liver, and some fibroid changes in the lungs. The brain, cord, and spinal nerve roots were normal. The articulations of the knee and fingers showed the characteristic changes. The cord and nerves were prepared in the same way as in the former case, and were examined with the following results: The cervical nerve roots and nerves of the arm were normal. The smaller nerve branches of the fingers all showed degeneration to a greater or less degree, as did also the filaments of the knee joints, and the sciatic nerves in the lower part of their course. The cord was normal.

In reviewing the first of these examples recorded by Mott and Tredgold, it appears to me to be most aptly designated by these authorities as chronic rheumatoid arthritis.

The widespread muscular atrophy, the character and extent of the distortion of the extremities, including the fingers and toes, the appearance of the skin of the hands, with its smoothness and cold surface temperature, and atrophy of the nails, when taken together, constitute a typical clinical picture of rheumatoid arthritis of long standing.

The one unusual feature consists in the presence of osteophytic outgrowths.

We have before noted the occasional appearance of bony proliferations in rheumatoid arthritis, and discussed their differentiation from those of osteo-arthritis. It seems to us, therefore, that their presence in this case is an epiphenomenon, one, moreover, which cannot be permitted to outweigh the general clinical aspect of the case, which speaks so emphatically for its rheumatoid nature.

Now, with regard to Triboulet and Thomas's case, the absence of any detailed description of the joint lesions renders it difficult, if not impossible, to exactly classify this case.

The history, the acute course run by the disease, and its comparatively early termination by death, strongly point to an infective disorder ; and, although it has been cited in connection with rheumatoid arthritis, I am inclined to think that it does not come within this category, but is an example of the so-called infectious arthritis. The same difficulty that besets us in dealing with Mott and Tredgold's case is again in evidence in relation to

Pitrès and Carrière's. In these examples, the acute stage of the malady has passed, leaving us with only the terminal results of the long-protracted disease. Had these cases come to autopsy in the early stages of the malady, it is quite possible that new bony formations would have been absent, and then there would have been no difficulty in classifying them as rheumatoid arthritis, into which category I have relegated them for reasons similar to those adduced in Mott and Tredgold's case.

VISCERAL CHANGES.

Cardiac and Vascular Lesions.—Endocardial and pericardial lesions have been noted by several observers.

Charcot and Cornil mention several instances ; but they are comparatively rare ; and, considering the eminently toxic nature of the affection, it is somewhat surprising that such have not been observed more often.

Dr. Katherine Clark[29] noticed general thickening of the arteries in three cases, in one of which the patient was only 24 years of age. In this latter, all the coats of the medium-sized arteries showed marked thickening, especially the tunica intima, which was thrown into folds, producing an irregular triangular shape in the lumen thereof. Similar changes were found in the smaller arteries affecting the tunica media and adventitia, and also the tunica adventitia of the veins.

Changes in the Liver.—The same observer found increased connective tissue in the portal tract, with thickening of the walls of the ducts and vessels, the fibrosis in one instance being very marked.

Changes in the Spleen.—In three cases thickenings of the walls of the central artery of the Malpighian body were present, being of the nature of a hyaline degeneration. The capsule and trabeculæ were also similarly affected.

Changes in the Pancreas.—Extensive increase of connective tissue throughout this organ has been noted in three cases, with degeneration of the parenchyma, hyaline thickening of the vessels and ducts, with proliferation of their lining epithelium.

Changes in the Ovary.—In one case, a girl of 24 years of age, total absence of germinal epithelium and follicles, with extensive fibrosis and hyaline thickening of the vessels, were noted.

Changes in the Kidney.—Extensive fibrosis of this organ has been noted, with hyaline degeneration of vessels and glomeruli.

Changes in the Skin.—In sections of this structure, atrophy of the epithelium glands and hair-follicles, with increase of fibrous tissue in the cutis vera, and hyaline degeneration of the deeper layers, have been noted, with also changes suggestive of chronic arteritis in the related vessels.

I am indebted to Dr. Katherine Clark for these observations on the morbid changes in the viscera of rheumatoid patients. As this observer suggests, it is difficult to account for the widespread arterial changes, except on the basis that they are due to chronic irritation by circulating toxins.

The alterations in the skin, this observer thought, were probably in part neurotrophic, partly vascular, in origin.

The changes in the viscera are, I think, especially interesting. Considering the scanty attention that has been paid to this aspect of the subject, the uniformity of the pathological changes in the three cases examined seems to indicate that further research along these lines might throw, as we have before indicated, considerable light on the pathogeny of this disease.

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CHAPTER VI.

PATHOGENY.

THE pathogeny of rheumatoid arthritis consists for the most part of a series of guesses and gaps. Owing to the scanty post-mortem evidence available it is largely composed of theories based upon the symptomatology and physical appearances of the disease and deductions drawn from the pathological data afforded us by diseases with which it appears to have analogies.

The two theories, the infective and the neural, still have their respective advocates, and neither seems able to overthrow the opposing view. As we saw when dealing with the bacteriology, it cannot be claimed that any specific organism is constantly associated with the disease. Moreover, careful observers, even when working upon the same lines of technique, have arrived at directly contradictory results ; some having been able to isolate organisms, while others have found the joint fluids sterile. Thus Odery Symes, after the examination of many examples, arrived at the conclusion that " in the majority of cases no organism can be detected, either in the blood or in the fluid of joints." Inasmuch as many students of the disease concur in this opinion, we may legitimately infer that the disease is not due to a specific organism with its nidus in the affected joints.

The absence of organisms, however, does not put out of court the infective theory of its causation, which is, indeed, strongly indicated by certain general features, viz., the previous history of infections, glandular and occasional splenic enlargement, etc. It does, however, suggest that the joint swellings may be the result of toxins derived from some remote site, possibly, as we have suggested, in many instances, the gastro-enteric tract.

Clinically, there is much to be said in favour of the disease being of the nature of a chronic toxæmia. The prolonged course of the malady, its remissions and exacerbations, with intervals of apyrexia alternating with pyrexial periods, are very significant of some intermittent discharge of toxins from a hidden focus into the general blood-stream.

Now, while this toxæmic theory does afford a reasonable explanation of many of the phenomena of the disease, there are other features which appear to indicate some participation on the part of the nervous system. Even the most ardent advocates of the infective theory seem unable to explain some, at any rate, of the collateral features of rheumatoid arthritis without invoking its aid. In like manner, those who uphold the neural view of its origin are hampered by their inability to explain on this basis those particular characteristics so indicative of some infective or toxic condition.

Being impressed with the disabilities that beset the purely neural or infective theories, I came to the conclusion that the varied phenomena of the affection were more adequately explained by regarding the condition as due to a cerebrospinal toxæmia; in other words, that the disease was of the nature of a trophoneurosis of infective or toxic origin.

It now seems apposite to note those particular clinical and pathological features of the disease, which have led so many strenuously to urge its primarily neural origin.

Taking the view, as the older authors did, that rheumatoid arthritis and osteo-arthritis were but expressions of one and the same pathological process, they were led, from the similarity displayed by the anatomical lesions of osteo-arthritis to the nervous arthropathies, to assign to arthritis deformans as a whole a similar neurotrophic character. As might be expected they found much in the clinical aspect of rheumatoid arthritis which tended to confirm them in the conclusions they had drawn from the pathological characters of osteo-arthritis.

Now, inasmuch as we believe these two conditions to be distinct, it is not permissible to include this similarity of osteo-arthritic lesions to those of Charcot's joints as part of our argument, in an attempt to assign a toxic trophoneurotic origin to rheumatoid arthritis. We shall therefore have to rely upon certain features peculiar to the latter to support this view as to its pathogeny, and the data that can be legitimately utilized would appear to be the following:—

1. The history of previous infectious disorders, the occasional

precedence, or co-existence, of gastro-intestinal and genito-urinary derangements, and localized foci of septic absorption.

2. The presence, for a variable period before the appearance of joint swellings, of symptoms which by analogy are best explicable on the basis of a chronic toxæmia, such as cramps and vasomotor and sensory phenomena.

3. The occasional onset of muscular atrophy before joint lesions are present; the incidence of atrophy in muscles remote from the affected articulations; the rapidity with which widely-generalized muscular atrophy may take place, too swiftly, indeed, to be accounted for by disuse, and quite out of proportion to the severity of the arthritic lesions.

4. The frequency with which marked exaggeration of the deep reflexes is present.

5. The tendency to a centripetal spread of the joint lesions from the periphery to the root of the limb; the ultimate symmetry of distribution obtained by the joint lesions, and certain collateral features; the tendency noted by McCrae, as well as myself, for the joint lesions to approximate to a segmental arrangement, coupled with a similar tendency to associated action on the part of certain superficial reflexes, such as the gluteal and plantar, which are governed by the same segment.

6. The ultimate dystrophic changes in the skin and subcutaneous tissue, as evidenced by atrophic changes, sensory and calorific disorders, with loss of hair and trophic alterations in the nails, early localized sweats, followed in the later stages by cessation of this function over the affected areas.

7. Finally, the resemblance of the muscular contractures present in the terminal stages to those met with in diseases of the central and peripheral nervous system.

Now, in reviewing the above characteristics, the fact that there are prodromal symptoms in rheumatoid arthritis constitutes to my mind an important argument in favour of the disease being a spinal toxæmia. These prodromal symptoms antedate the appearance of joint swellings by an indefinite period, a period so long that it precludes the possibility of their being reflexly related to the joint swellings. Again, in the early stages of rheumatoid arthritis it is significant that while the joint-changes may be confined to the fingers of one hand, these prodromal features, as evidenced by vasomotor phenomena, etc., may already be present in the opposite hand, hitherto unaffected by the joint lesions. This would appear to suggest that the joint swellings are not the beginning of the disease, but are rather the final or end manifestation of the previously long-existing toxæmia, of which they are a further elaboration. Collectively looked at, these prodromal signs all possess one feature in common, i.e., they appear to stand in intimate relationship to the joint troubles, inasmuch as their manifestations are confined to, or are more marked in, those areas in which periarticular swellings subsequently develop.

As regards their relative incidence, or absence in any given case, they vary greatly. One or other, the vasomotor symptoms, the cramps, or sensory disturbances, may be absent; and of those present, one usually stands out more prominently than its fellows; and thus we may get a condition clinically comparable to Raynaud's disease, or one closely resembling a paroxysm of tetany.

Now, if in our attempt to grasp the significance of this variation in the prodromal features, we take a paroxysm of tetany with its attendant vasomotor phenomena, and compare it with a paroxysm of Raynaud's disease with its associated muscular cramps—for they certainly occur in this disease—the question presents itself, Is there so much difference clinically?

Does not the distinction lie mainly in the varying prominence of certain symptoms, and those symptoms common to both? Thus in tetany, the muscular cramps dominatê and colour the picture, pushing the accompanying, though less obtrusive, vasomotor phenomena into the background; while in Raynaud's disease the vasomotor phenomena stand out in bold relief, and in their turn the muscular cramps lapse into comparative obscurity. May we not see in these superficial differences an indication of a profound resemblance in the two diseases? Are they not both probably dependent on some underlying cerebrospinal toxæmia? Such has been acknowledged by many to be the case in tetany; and signs are not wanting that such may be the case in at any rate some examples of Raynaud's disease. Note its incidence suddenly after intestinal obstruction, likewise the occurrence of "dead" and "white" fingers in the course of pernicious anæmia. I have gone into this detail, because in the same way the prodromal features of rheumatoid arthritis are mingled in varying degrees, one or other predominating; hence, reasoning by analogy, it seems permissible to allot to them a similar toxic origin.

Again, taking these three diseases from the point of view of their etiology, no one who examines into that of rheumatoid arthritis can help being struck by its varied nature. Acute specific diseases, gastro-intestinal disturbances, genito-urinary disorders, etc., have all been credited with some relationship to the affection.

Similarly, in tetany, diversity is a striking feature of its etiology—gastro-intestinal disorders, diseases of the thyroid, pregnancy, puerperal conditions, not to mention acute specific fevers; and in the same manner Raynaud's disease is affiliated to numerous widely-differing diseases, amongst them pernicious anæmia, chronic Bright's disease, malaria, and so forth. Does not this manifold etiology seem to suggest that each of these three diseases is not a clinical entity, but rather a symptom-complex, owing its production to a variety of diseases, the residual toxins of which, acting on the nervous system, evoke their particular clinical features?

Hitherto we have been comparing the premonitory symptoms of rheumatoid arthritis with those of Raynaud's disease on the one hand, and those of tetany on the other. It is interesting, therefore, to find, in addition to the resemblance above alluded to, this clinical fact common to all, that each may be attended by periarticular swellings. Thus Risien Russell,[1] in his article on tetany, states that joint-swellings may supervene in this disorder, and that such are not of a "rheumatic" nature; but are to be ascribed to "trophic and vasomotor disturbances of innervation." Again, Hutchinson[2] relates the association of Raynaud's disease with what he terms "last-joint arthritis"; while Southey and Weiss have reported cases in which the larger joints are involved. Weiss attributed these coincident joint-affections to some temporary derangement of the hypothetical joint-centres in the cord, regarding them as mild forms of arthropathy.

Now, if we are content in tetany, likewise in Raynaud's disease, to assign a trophoneurotic origin to the joint-swellings that occasionally supervene in their course, why may we not admit the possibility of such an origin in the case of the periarticular swellings of rheumatoid arthritis. It seems to me that given any case of tetany or Raynaud's disease with periarticular swellings, in both cases situated in the smaller joints, the sites of election in rheumatoid arthritis, it would be very difficult to differentiate clinically between them and a case of early rheumatoid arthritis. I have seen such cases which might have indifferently been labelled Raynaud's disease with periarticular swellings, or rheumatoid arthritis with marked vasomotor phenomena. Similarly, in others where periarticular swellings are accompanied by marked muscular cramps, it is difficult to exclude tetany: all the more so when there is present an intercurrent malady, gastric ulcer, or other gastro-intestinal disorder known to be etiologically related to the latter. These difficulties naturally only arise when dealing with early rheumatoid arthritis, in which the joint swellings are of recent date, and confined to one or more of the small joints of the hands.

Consideration of the foregoing points of similarity in these three diseases has led me to question whether they may not under some conditions gradate insensibly into each other as members of one family group, owning one common parent origin, a cerebrospinal toxæmia. It seems to me that only in this way can we explain this overlapping of these three diseases, this erratic trenching of one upon the clinical territory of the other, this similarity of clinical expression. It seems to justify one in claiming a kinship for these three affections, a common mode of genesis which provides that however diverse the etiology or the sources of the toxæmia, they shall exert their influence *viâ* one controlling medium, the nervous system: hence the strong family likeness running through the three diseases.

In continuation, when we come to note the clinical facies of rheumatoid arthritis when mature we find, at any rate in some of the cases, a striking resemblance to some diseases of central nervous origin, as evidenced by symmetrical joint lesions, widespread and intense atrophy, increased reflexes and trophic changes, in short, a clinical picture for which no parallel can be found in any purely arthritic disorder. Thus, in no other disease do we get such a combination of symptoms as one may see in the hand of a rheumatoid patient, with its spindle joints, its recurring syncopal and asphyxial changes, its localized sweats and muscular atrophy. Many, like Ord, have considered that this combination constitutes a dystrophy analogous to progressive muscular atrophy; but to me it seems more comparable with syringomyelia, in which disease the hand may be the seat of multiple arthropathies, attended by muscular atrophy, vasomotor changes, and secretory disturbances. Presumably Teissier[3] also notes a superficial resemblance between these two diseases, inasmuch as when discussing the differential diagnosis of rheumatoid arthritis he deems it necessary to exclude in detail cheiromégalie syringomélique and Morvan's disease. It need hardly be mentioned that I am merely drawing attention to a certain general superficial resemblance that rheumatoid arthritis has to this disease despite its very diverse nature.

Again, the symmetrical distribution of the articular lesions and other phenomena has been cited by many authors as an indication of the central origin of the disease ; but there has been of late an attempt to depose the premier tissues of the body, the nervous structures, from their high estate, and to contend that symmetry of lesions is a common characteristic of many disorders in which the all-pervading medium, the blood, is contaminated. There is nothing new about this latter suggestion, inasmuch as this view of the origin of the symmetrical diseases was long since promulgated by Sir James Paget and Dr. William Budd, who contended that the materies morbi entered into combination with those tissues for which it apparently had a chemical affinity ; and those parts of the tissues which occupy symmetrical positions on the opposite sides of the body being presumably identical in their composition, the outward symmetry is thus explained, being merely the reflection of a deeper and more subtle identity of chemical constitution with its associated affinity for the morbid material. If we substitute the word toxin for the phrase materies morbi we have the more modern view.[1]

It is, however, interesting to record that subsequently to his paper on "Symmetrical Diseases," published in the *Medico-Chirurgical Transactions* for 1842, Sir James Paget came to the conclusion that it is "more probable that the symmetrical distribution of diseases on corresponding portions of the upper and lower extremities should depend on their central nervous relation than on any assumed identity of chemical constitution."

But a limit must be set to the bounds of conjecture, and having regard to our ignorance of the more intimate selective chemical affinities of toxins for the various bodily tissues, and the impossibility of ruling out all influence exerted by the central nervous system in determining the character and sites of the lesions, too much stress must not be laid on either the chemical or the neural interpretation of symmetry as a phenomenon in disease, the truth lying probably in a combination of these two views. It may, however, be claimed that the centripetal spread of the lesions, and their occasional segmental distribution in the joints of the hands, with the curious harmony that prevails between the gluteal and plantar reflexes, are rather suggestive of primary spinal cord involvement, and indirectly carry with them the inference that the symmetry of rheumatoid arthritis is possibly central in origin.

The muscular atrophy, as we have seen, presents certain peculiarities of distribution which cannot be satisfactorily accounted for on the basis of a purely reflex arthritic muscular atrophy. Even those who find in it the most adequate explanation of the muscular atrophy seem dubious as to its applicability to all cases, inasmuch as instances occur in which atrophy may not only precede the joint lesions, but may be present in muscles remote from the affected articulations.

But however satisfactory and adequate this explanation of the muscular atrophy by reflex irritation may be deemed by some, it still leaves us with a number of collateral features—pigmentary, trophic, vasomotor, and sensory disturbances—still unaccounted for; and inasmuch as these additional collateral features are as essentially a part of rheumatoid arthritis as the muscular atrophy, it is necessary to look for some more comprehensive theory of its pathogeny.

Now, whether they be regarded as primary or secondary, it is, at any rate, certain that changes have been found in the anterior cornua, as evidenced by Folli,[4] Mott,[5] and Bannatyne;[6] and their similarity in Mott's case to those obtaining in progressive muscular atrophy and amyotrophic lateral sclerosis has already been commented on. Again, the same provisional interpretation, whether primary or secondary, applies to those lesions of the peripheral nerves described by Pitrès and Vailliard,[7] and later by Pitrès and Carrière.[8] How far these lesions, whether central or peripheral in distribution, are responsible for the articular changes, muscular atrophies, and trophic disturbances, must be left open. But, at least, these changes do support the possibility that the condition of rheumatoid arthritis may be due to the actions of toxins on the central nervous system. It must be admitted that such changes have frequently been found absent; but this does not exclude the possibility of some dynamic change having taken place in the governing nerve cells, and we have every indication clinically that such may be the case, for not only do we find the vasomotor and other phenomena in the early stages of the disease transient and periodic; but even the joint swellings may rise and wane from time to time, and this ephemeral tendency is probably the outcome of similar changes in the nervous system consequent on variations in the amount of toxic material carried to the cells by the contaminated blood-stream.

Moreover, this frequent lack of coarse changes in the nerve-centres is quite in keeping with what we see in other chronic toxæmias. In tetany, for example, while widespread changes have been found in the meninges, cord substance, and peripheral nerves, in severe cases, in others, of slighter intensity and more brief duration, such have been absent. Again, apart from tetany, rheumatoid arthritis approximates to other chronic toxæmias in that posterior sclerosis which has been found occasionally in pernicious anæmia, diabetes, and Addison's disease, and ergotism has also been met with in rheumatoid arthritis; and thus we see that, not only clinically but pathologically, it tends to identify itself with disease of this nature. The inconstancy with which these cord-lesions are found resolves itself apparently into a question of the duration of the disease, the toxins giving rise at first only to dynamic changes, and later to degenerative alterations.

These changes in the cord-substance and peripheral nerves do not exhaust the morbid material on which the central origin of rheumatoid arthritis is based, for Teissier, in a certain number of post-mortems performed by himself, ascertained the existence of diffuse patches of spinal meningitis of variable size, compressing in some instances the spinal nerve roots; and he further remarks that in the course of overlooking the post-mortem records of cases by other authors he has often incidentally noted that plaques of spinal meningitis were observed. In this connection he draws attention to a case recorded by Touche, in which the dura mater in the neighbourhood of the posterior roots in the cervical region was quadrupled in thickness. He refers also to a case by Leri, in which lesions of the same nature were found. Teissier, in discussing the pathogeny, is inclined to the view that the disease is of the nature of a trophoneurosis of toxic or infective origin; and that in their endeavour to ascertain the site of the causal lesion pathologists should examine systematically not only the cord but its enveloping membranes. This compression of the posterior nerve roots by chronic spinal meningitis seems to me to afford a very reasonable explanation of the persistent neuralgic pains that characterize certain forms of rheumatoid arthritis; and, incidentally, it may possibly account for the benefit which undoubtedly follows counter-irritation applied in the neighbourhood of the cervical and lumbar enlargements.

Passing now to the consideration of the features of the disease in its terminal stages, one notes the prominence of the muscular contractures, and we have before alluded to the striking resemblance of the distortions produced to those met with in diseases of the central nervous system in which joint lesions are not present, such as atrophy of the interossei, congenital atrophy of the brain, certain hemiplegias, and paralysis agitans. Teissier, commenting on the striking analogy between the deformities of rheumatoid arthritis and those produced in diseases of this class, remarks that it is sometimes difficult to differentiate them.

He illustrates this by way of an example, citing a case of cerebral diplegia in Marie's clinic where the deformities of the hand resembled those peculiar to that variety described by Charcot in rheumatoid arthritis as the type of flexion, and in which skiagraphy showed that similar destructive changes in the cartilages and articular ends of the bone were taking place in those articulations most deformed.

The grave changes found in the skin, more particularly that of the hands and fingers in the later stages, their shrunken, cold, and bluish-white appearance, are all suggestive of some grave trophic disturbance, and, as we have before suggested, may be due to the constantly recurring circulatory disturbances. This possibility is supported by the fact that the fingers, as the result of long-continued Raynaud's disease, may present the same atrophied, tapering, parchment-like appearance. It may be mentioned incidentally that Raynaud's hypothesis as to the pathology of the disease described by him was that it should be ascribed to an unduly irritable condition of the vasomotor centres.

In reviewing the foregoing remarks on the pathogeny of rheumatoid arthritis, we have endeavoured to show in the first place that the premonitory symptoms are such as can best be explained on the basis of a chronic toxæmia; similarly, when viewing the disease in its maturity, that the complex syndrome presented of articular lesions, vasomotor phenomena, sensory changes, and muscular atrophies, are most easily explicable on the same hypothesis, namely, a toxic invasion of the nervous centres governing these various structures. In continuation we have also shown that the ultimate or terminal facies of the disease presents characteristics similar to those met with in disorders of central origin, and which in their character are such as might very reasonably be ascribed to degenerative changes in the cord, the results of protracted toxæmia.

As regards the joint lesions themselves, we have already pointed out that authorities differ as to the interpretation to be placed on the histological appearances presented. Some, like Painter, consider that they are eminently such as might be explained on a neurotrophic basis; while others maintain that they are best explicable as the effect of an inflammatory process. Pending the final settlement of this vexed question as to the nature of the joint lesions, it seems to me that the two most likely hypotheses are the following:—

Firstly, that the disease in its manifold complexity is secondary to a chronic toxæmia, which exerts its influence *viâ* the nervous system; the articular lesions and collateral features being due to changes primarily of a dynamic nature, and later of a degenerative type induced by the toxins. Or, else, that the toxins associated with the disease possibly have some selective affinity for the synovial tissues, creating a toxigenic synovitis ; and that the collateral features are due to toxins acting on the nerve centres, the two changes proceeding *pari passu.* I have already alluded to the fact that Teissier inclines to the view that it is of the nature of a toxic trophoneurosis ; and I may add that Macalister, referring more particularly to those cases of rheumatoid arthritis attended by marked vasomotor phenomena, maintains that the condition is due to a toxin, which acting through the nervous system produces vascular spasm, which in some obscure way determines the altered nutrition and ultimate destruction of the joint structures.

McCrae[9] also, though strongly in favour of the infective origin, was led by the segmental distribution of the lesions and the curious associated alteration in the segmentally-related reflexes to suggest that the chronic cases of the polyarticular type of arthritis deformans with their nervous manifestations might possibly belong to the class of chronic toxæmias. Latham,[10] as is well known, considers the disease due to changes in the nerve centres ; the articular lesions, in his opinion, closely resembling the joint changes that follow cord or nerve lesions.

Stengel[11] considers that the history of cases of rheumatoid arthritis shows plainly the operation of influences, the result of which must be deterioration of nerve force and vitality ; and that the clinical features and course of the disease are such as are quite compatible with the view that primary changes in the cord, either central or peripheral in site, constitute the true essence of the pathology of this disease. He lays stress upon the peculiar type of the articular lesions as being painless, progressive, and dystrophic in character, and attended by evident alterations in the skin of affected parts. He does not consider the purely microbic theory merits serious consideration. Goldthwait,[12] speaking of atrophic arthritis, considers that some of the features strongly suggest some trophic change secondary to a nerve lesion.

In conclusion, the pathogeny of rheumatoid arthritis is obviously very largely speculative, the changes in the nerve centres being so inconstant as to preclude any dogmatic statement as to its causation; but the scantiness of the data at our disposal does not absolve us from an attempt to explain as best we can the varied phenomena of the disease, in the hope that we may anticipate future pathological findings.

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Chapter VII.

PREMONITORY SYMPTOMS.

That it is of prime importance that we should at the earliest possible period appreciate the true nature of any oncoming malady is sufficiently obvious, and in no disease are the consequences of inaccurate or tardy diagnosis so deplorable and irremediable as in rheumatoid arthritis, a disease so difficult of amelioration, so defiant of cure. In its advanced stages the clinical picture presented is so characteristic that he who runs may read, but at this belated period of its life history there is but little "kudos" attaching to its recognition; still less is there any utility from a therapeutic point of view.

Realising, therefore, the intractable nature of the disorder, all writers unite in emphasizing the necessity of early diagnosis. That Sydenham[1] was alive to the lamentable consequences of delay or inaccuracy in this connection may be gathered from his sympathetic allusions to the tortures endured by its victims when improperly treated, and that Haygarth[2] felt strongly on the point is apparent from the tenor of the words in which he introduced the disease to the notice of the physicians of his day. "This faithful picture drawn from nature is here exhibited to excite the compassion and exertion of my professional brethren to prevent, if possible, so distressful a malady at *its commencement.*"

Coming to the same conclusion as Haygarth, that the ideal treatment of rheumatoid disease is, if possible, to prevent it, or falling short of that to recognize and treat its earliest manifestations, more modern students of the disease, notably Fuller, Garrod, and Spender, have endeavoured to trace the insidious beginnings of the malady. As a result, these authorities have noted certain symptoms—symptoms, moreover, not necessarily arthritic—which, preceding the joint-troubles, as they not infrequently do, may be legitimately termed premonitory. It is with a consideration of these prodromata that this present chapter is concerned, and for the better convenience of description I shall consider them under various headings: those referable to the vasomotor system; subsequently those pertaining to the muscular and sensory systems. As I have before said, the divisions are purely arbitrary, and although I shall adhere pretty closely to them when discussing the vasomotor and motor troubles, I shall towards the end of the section devoted to sensory disorders depart from this more or less artificial separation, and shall there treat them as they appear to us in actual clinical work, namely, mingled with each other in varying proportion in different examples of the disease, but nevertheless one or other of them predominating.

VASOMOTOR GROUP OF PREMONITORY SYMPTOMS.

Attention was first drawn to these phenomena by Kent Spender, who in his inimitable manner described "the purple-blue hands, frigid even on a hot summer's day, so commonly to be seen amongst rheumatoid patients, and their mottled fingers, so dark in tint, that they reminded him of the vagaries of Raynaud's disease." In the same manner, when first brought into contact with a number of such patients at the Bath Mineral Water Hospital, I was struck by the frequency with which they complained of "dead" fingers. Others, again, more observant, would remark that their fingers were always changing colour: at one time they would be dead white, at another purple or blue. On going further into the matter, I found that many of these rheumatoid patients were indeed suffering from frequent attacks of local syncope and local asphyxia. Their similarity to those of Raynaud's disease at once suggested itself, and though at first I hesitated to apply the term to them, further observation convinced me that clinically they were indistinguishable from those avowedly of the type described by Raynaud, for the following reasons:—

1. They fall easily into the three classical divisions: local syncope, local asphyxia, and symmetrical gangrene.
2. They are definitely paroxysmal in their mode of incidence.
3. Their sites of election are the extremities, the hands and feet, the tips of nose and ears, and very rarely the genitals.
4. The attacks, whether of local syncope or local asphyxia, are symmetrically distributed.

Now, as we know, in Raynaud's disease the vasomotor phenomena present infinite gradations in intensity and extent; so in the same manner do we find a similar capriciousness characterizing these manifestations as they occur in the subjects of rheumatoid arthritis. Thus, while on the one hand in many rheumatoid patients these signs of local syncope and local asphyxia are slight, and by reason of their ephemeral nature readily overlooked, so on the other hand in isolated instances we find them widespread and intense, legitimately comparable to the more aggravated examples of Raynaud's disease. It is obvious that it is only in the light afforded us by the more severe types of Raynaud's disease that we are able to grasp the full significance of the more trivial ones, and it was for this reason that I some years ago recorded in the *Edinburgh Medical Journal* some instances of rheumatoid arthritis, in which the vasomotor disturbances were unusually severe and widespread. The fact, therefore, that such intense phenomena of this character may occur in this connection, naturally to my mind suggested that the more common and less intense forms were but minor manifestations of the same nature, differing in degree, but not in kind. Moreover, in order to further justify my contention that there is no essential difference between the vasomotor phenomena which occur in rheumatoid arthritis, and those of Raynaud's disease, I sought for the rarer manifestations of the latter in the subjects of rheumatoid arthritis, and the results obtained, to which I shall allude later on, support my claim that they are, clinically at all events, indistinguishable.

Now as regards the relationship of these vasomotor changes to the joint swellings.

The subjects of rheumatoid arthritis may have suffered for years from local syncopes and local asphyxias before their joint swellings appeared. Not uncommonly, also, we get a history in such patients of other paroxysmal neuroses, notably migraine and more rarely asthma. In other instances no such long interval exists between the onset of local syncopes and asphyxias and the subsequent appearance of periarticular swellings. Not infrequently the vasomotor phenomena may develop almost suddenly in sequence to some infective or toxic condition such as influenza, sore-throat, and so on, to be followed in a few days or weeks by the development of so-called spindle-shaped joints. Thus, going more into detail, the first symptom may be a condition of local syncope or asphyxia in the ring and middle fingers of one hand, accompanied as a rule by a mild degree of spasm of the flexor muscles of those fingers : in some instances the muscular spasm is restricted to those fingers, but, as a rule, the other digits usually participate in a minor degree.

The vasomotor paroxysms may last for ten or twenty minutes before relaxation sets in. During the cycle the surface temperature of the affected area is lowered, with some transient sensory modification of the related skin, such as diminished tactile sensibility, or, what is more easily ascertained, diminished sensibility to painful stimuli. In addition, there is slight œdema, and the whole attack terminates in sweating over the affected area. In rare instances an extension of the vasomotor phenomena up to the forearm may result, and the path followed seems to me to indicate a spinal rather than a peripheral nerve distribution. In the case of the ring and middle fingers, this extension takes place over the dorsum of the hand and forearm, adhering more or less accurately to the eighth cervical territory (Head). The segmental sweating also confines itself to the same area, and there is the same sensory modification as occurs in the fingers.

In some cases there is in addition localized sweating in the centre of the palm over an area extending from the base of the ring and middle fingers to the wrist, governed also by the same segment (the 8th cervical). The increased tonicity of the flexors of the wrist and fingers, as evidenced by their spasm and brisk response to percussion, seems to point the same way, inasmuch as they are innervated by the same segment. Now should joint swellings be imminent a notable increase takes place in the extent, frequency, and severity of the vasomotor paroxysms. As a result of their more rapid incidence, the œdema, at first no more marked over the joint than the rest of the finger, eventually, by reason of the lax condition of the skin over the articulation, becomes more obvious at this site, and a gradual filling up of the redundant skin ensues. As the result of careful study of instances in which the periarticular swellings actually developed under my own observation, I cannot help thinking that the more superficial characters of the spindle swelling, namely, the changes in the skin and subcutaneous tissue, are the result of the, at this time, rapidly recurring vasomotor paroxysms, the œdema incidental to these vasomotor symptoms probably assisting in the distention of the skin and obliteration of wrinkles. After this formation, the spindle joint preserves in the interparoxysmal periods the more or less constant waxy-white or semi-asphyxial tint noted by so many observers. I have before stated that a considerable interval of time may elapse between the onset of the vasomotor phenomena and the appearance of periarticular swellings, but supposing the latter to have appeared and subsided, as not infrequently happens in the early days of rheumatoid arthritis, then, when owing to some fresh infection an exacerbation of the vasomotor phenomena takes place, the periarticular swellings form much more quickly, it may be even in two or three days. In brief, we have to my mind here a series of Raynaud-like paroxysms which eventually may have the added phenomena of spindle or periarticular swellings.

I will now cite a few examples.

H. D., æt. 20; female. One month after a severe sore throat noticed a "tingling" sensation over the middle of the forearm both anteriorly and posteriorly, the "tingling" extending to the tips of the ring and middle fingers. At the same time the affected fingers turned "dead white" in colour. The attacks lasted from fifteen to twenty minutes, and were especially severe at night, keeping her awake. Coincidently the fingers were flexed with a cramp-like sensation. Within three weeks the mid-phalangeal joints of the middle and ring fingers became spindle-shaped, and afterwards the knuckles of the same fingers became enlarged. The same sequence of events happened in both hands, and it is interesting to note that nodules appeared shortly afterwards in both extensor and flexor tendons of both hands. The joints afterwards became more widely affected.

E. C., female, æt. 36; single. She had suffered for some years with symptoms pointing to gastric ulcer, with hæmorrhage from the stomach on several occasions. During all these years she had suffered from syncopal attacks in the fingers. The last gastric hæmorrhage occurred a month previously to the onset of joint swellings, and during the intervening month a great increase in the frequency and severity of the syncopal seizures was noted.

R. M., female, æt. 21. Four years ago she suffered with anæmia, also symptoms of gastric ulcer with hæmatemesis. The next symptom noticed was that her big toe used to go "blue and white often," and then swelled. The same sequence was observed in the opposite big toe; afterwards both ankles swelled, also knees. Her hands went through the same changes —local asphyxias followed by swelling of joints. She at this time noticed that she "trembled" a great deal. About twelve months ago she noticed her collars getting "too tight" and that she suffered with "palpitation," "feelings of heat," and causeless diarrhœa.

For the relief of her joint condition she was sent to the Mineral Water Hospital, and presented, in addition to rheumatoid arthritis, the features of Raynaud's disease and Graves's disease. Her thyroid gland was enlarged, her pulse-rate was 100, with slight general tremor, proptosis, with Von Graefe's sign and highly arched eyebrows.

Macalister,[3] of Liverpool, more recently but quite independently of myself, has also noted this association of Raynaud's disease and rheumatoid arthritis, and also lays stress on the fact that these attacks of local syncope and asphyxia are "not infrequently very early symptoms of rheumatoid arthritis, and exist long before the joint troubles begin." Douglas Stanley has also noted this same sequence of Raynaud's disease followed by rheumatoid arthritis in a young child.

Spender,[4] on the other hand, in his book on early symptoms, does not allude to the important fact that these symptoms often precede joint swellings.

Before closing my remarks on these phenomena, I would lay stress on the following facts :—

1. That these so-called "dead blue-and-white" fingers, etc., are very common forerunners of rheumatoid arthritis.

2. Their manifestations are usually more severe in, if not actually confined to, those digits which afterwards become the seat of periarticular swellings.

3. Any notable increase in the severity of the vasomotor spasms, both as regards frequency and extent, should arouse suspicion, as such exacerbations often denote the speedy oncoming of joint swellings.

MOTOR GROUP OF PREMONITORY SYMPTOMS.

These readily fall under three headings: Muscular spasm, muscular weakness, muscular wasting.

Both Charcot and Trousseau noted the frequent occurrence of muscular spasms in the subjects of rheumatoid arthritis. The former[5] believed them to be of reflex arthritic origin, but Trousseau,[6] on the contrary, thought it more rational to regard them as "a manifestation of the general local state," a manifestation which may be absent, but which is sometimes a predominating feature of the disease. He refused to recognize them as a consequence of the arthritis, inasmuch as he had observed that in some cases the muscular spasms preceded the joint lesions.

Many writers have since confirmed the observation of these two illustrious French physicians, that muscular spasms occur very frequently in the course of rheumatoid arthritis, but, on the other hand, few have supported one of them, Trousseau, in his plea that the muscular spasms sometimes preceded the joint lesions. In an article communicated to the *Edinburgh Medical Journal,* in 1903, I drew attention to the fact, and this was reinforced by Macalister in an address on the subject delivered before the Liverpool Medical Society, in which he emphasized the fact that these muscular cramps are very common initial symptoms of the disease, and that they have not received the recognition they deserve. Teissier,[7] also, in his recent work on the subject, emphasizes the importance of these muscular spasms as premonitory indications of the oncoming of rheumatoid arthritis. These muscular spasms may be either continuous or paroxysmal, and like the vasomotor phenomena tend to be symmetrical. They are often intensely painful, and in severe cases may continue almost without remission for hours.

They occur most frequently in the hands and feet, but may in severe cases involve the muscles of the arms, those of the thighs and calves, and in rare cases those of the head and neck.

In the hands flexor spasm is more common; the hand may be tightly clenched, so much so that the imprint of the nails can be seen upon the palms. I have known a mother insert the handle of a spoon to lever open the hand of her child thus afflicted. In other cases, the patients on waking from sleep may find their hands half closed, an approximation to the conical shape, and they resort to friction, soaking in hot water, etc., in order to relax the spasm. Again, in some, the flexor spasm ensues when the patient grasps some object, and the sufferer may be quite unable to relax her grip. As an example, I may quote the case of a lady who, seizing the handle of her telephone, was unable to release herself, and had to call her maid to disengage her hand. She suffered much in this way, and shortly developed joint swellings. Again, the wrist may be flexed and the hand adducted, a forecast of the ulnar deviation so characteristic a deformity of the disease in a later period. To a certain extent, the character of the spasm gives us an indication as to which joints are likely to be involved first; for example, if the flexor spasm of the fingers predominate, the joints of the ring and middle fingers will probably first show signs of swelling.

If the fingers are rigidly extended—and such cases do occur, though much more rarely—then the joints of the thumb and index finger should be closely examined for signs of enlargement. In more severe cases the spasm may be widespread, involving the whole upper limb; then the arm may be closely adducted, the elbow flexed and the forearm held midway between pronation and supination. In such cases the bicipital muscles are strikingly resistant and hard, and they appear to be swollen, and I have seen the overlying skin œdematous. This complaint of severe and extensive spasm may last for days with slight remissions, and is very likely to be confused with muscular rheumatism, but the attitude assumed by the patient is the one commonly adopted when rheumatoid disease in an acute form is attacking or is about to attack the radio-ulnar articulations, superior and inferior. Spasm of the muscles of the head and neck, as Spender pointed out, may be an early symptom of affection of the cervical vertebræ.

In the lower extremities the same phenomena may present themselves: the toes may be painfully flexed and drawn to the fibular side, and the painful irregular contraction of the plantar muscles may give the patient the sensation of "treading on marbles." More rarely the anterior tibial and calf muscles may be the seat of these "cramps," and even those of the thigh may be involved, particularly the hamstring group.

The malpositions produced by these muscular spasms which obtain during the period of invasion are, as Teissier points out, temporary in character, the deformity undergoing gradual correction during the interparoxysmal periods, and only by their constant repetition do the deformities become permanent.

MUSCULAR WEAKNESS.

Loss of muscular power, sudden in onset, fleeting in duration, is, as Spender noted, often a forerunner of rheumatoid disease in a limb. Such sudden, unaccountable weakness may betray itself by the unconscious slipping of articles from the grip, or may be by an oft-recurring, although transient, difficulty in picking up small objects. Such patients complain that their hands have no "use" in them, or that they will not "hold," and are often haunted by the fear of oncoming paralysis. In some cases the "weakness" appears suddenly during the performance of the more highly specialized movements, such as letter writing and piano playing, and the wrist is then regarded as the seat of the trouble, and in order to guard against any future surprises the joint is bandaged or recourse is had to artificial supports, binding with handkerchiefs and so forth. Another very frequent complaint made by such patients is that they cannot turn door-handles or taps, or take corks out of bottles. This is a most significant type of muscular weakness, as early diminution of the power to supinate is one of the most striking features of the disease. In all such cases the mobility of the forearm in this respect should be investigated, noting especially if any creaking is present in the superior radio-ulnar articulation. We have already pointed out that severe muscular spasm in the more acute types of the disease may precede or accompany involvement of the radio-ulnar articulation, but the same result may be attained insidiously : hence the importance of not dismissing cursorily the above disabilities when complained of by patients. Moreover, it is important in this connection to remember that this limitation of the power to supinate may occur long before flexion of the wrist and elbow joints becomes painful or restricted.

This early and insidious affection of the radio-ulnar articulations has not received the attention it merits. The superior articulation being very superficial is easily examined, and the information gained will often establish the rheumatoid nature of obscure trouble referred to the wrist joint. Recently I was consulted by a lady supposed to be suffering from a sprained wrist, the forearm and hand having been kept at rest by an anterior splint. On examination, I found some puffiness of the wrist, but on gentle rotation I was able to detect definite creaking in the superior radio-ulnar articulation. More rarely this sudden loss of power may be more widespread, affecting the whole of the upper limb, or may be both, as happened in a case under the care of Preston King. The patient, a man going forth to work one morning as usual, suddenly lost power in both arms. A few days later the joints began to be affected, and six months from the onset of the attack he was admitted into hospital presenting all the features of a well-marked case of rheumatoid arthritis.

Senator also alludes to a "sense of lassitude" in the joints as a premonitory symptom. Similarly, in the lower extremities one meets with examples of this sudden loss of power, and I have known a patient drop to the ground when overtaken by such a seizure. Spender also noted that patients with no conspicuous atrophy might feel as if all the strength had gone out of their legs, and that such sensations were transient and usually occurred during walking. Weakness of the ankles is not infrequently complained of, and oft-recurring twisting of these joints beneath them may have been the first symptom to attract the patient's notice.

MUSCULAR WASTING.

Actual wasting of the muscles of a limb may precede the appearance of joint swellings. Ord was, I believe, the first to point out this interesting and important fact, and in a published letter to Kent Spender states definitely, "I have seen the muscular wasting precede the osteo-arthritis." Spender again on this point says, "Sometimes the muscular wasting is distinctly in advance." Lorimer, of Buxton, also has noted a case where muscular wasting preceded the arthritis, while Lane and Griffiths state that muscular atrophy very frequently anticipates the joint changes. From the study of asymmetrical cases of the affection, I was led to the conclusion that while muscular wasting usually makes its appearance concurrently with the joint trouble, yet in isolated instances it may precede it, as evidenced by the fact that although the arthritic mischief might be confined to one extremity, the muscular atrophy was quite as marked in the opposite and apparently unaffected limb. Again, even when the trouble is localized, for example to one wrist joint, detailed examination not infrequently reveals the presence of marked wasting in muscles remote, such as those of the shoulder girdle, and of those more particularly the supra- and infra-spinati.

Similarly, when the disease is restricted to one foot, affecting, it may be, the mid-tarsal or ankle joint, we can often detect wasting and fibrillary twitching of the gluteal muscles on the same side, in addition to atrophy of muscles especially related to these joints, the anterior tibial, peroneal and extensors of the foot, and intrinsic plantar muscles. Here the evidence is more strictly in favour of the muscular atrophies being of central origin, inasmuch as we know that the glutei and the above-mentioned group of muscles all own allegiance to the same segments, namely, the second and third sacral of Head. The fact that such remote muscular atrophies may be present even when the joint swellings are peripherally localized is too often overlooked, owing to lack of detailed examination of the whole of the affected limb, and thus this substantial aid to diagnosis fails to be utilized.

SENSORY GROUP OF PREMONITORY SYMPTOMS.

Paræsthesiæ of all sorts may precede by weeks or months the onset of rheumatoid changes in the joints of the limb, tingling and numbness, "pins and needles," sensations of stinging and soreness, etc. Such sensory perversions are usually confined to the hands and feet, but in some cases they may ascend the limbs. Occasionally these abnormal sensations are evoked by peripheral stimulation; for example, a stream of "pins and needles" may extend up the arm when the fingers are brought into contact with any object, and I have known a similar nervous discharge occur whenever the foot touched the ground.

Howard[8] and Homolle[9] were the first to draw attention to these disturbances of sensation, and the former was struck by their resemblance to those met with in affections of the central nervous system. Garrod has also contributed examples of a similar nature, likewise Forsbrooke, and Lane and Griffiths. Lyman and others have also noted the frequency of these premonitory sensory aberrations.

Passing now to the consideration of pain as a premonitory symptom: "Nodular rheumatism, insidious in its beginnings, declares itself by pains," thus wrote Trousseau in his clinical lectures, adding that the pains were less characterized by violence than by constancy, and that a variable period may elapse before they are followed by joint swellings. He carefully distinguishes between the pains before alluded to, which are dependent upon muscular spasms, and these now under consideration, which are usually referred to the articulations. These recurring articular pains may be very acute, often strictly localized to a small area, and are frequently described by patients as being of a "gnawing" character. In my experience, the dorsum of the wrist, also that of the foot, are favourite sites, and in the case of the latter the pain is more often situated over the mid-tarsal joint. Spender attached great significance to persistent pain in the muscles of the ball of the thumb, and in the case of a girl under my care, the first symptom was the onset one night of severe gnawing pain at this site; it persisted for four days, and the opposite thumb suffered in exactly the same way. In the shoulder, the pain is usually referred to the interior of the articulation, but it may be of the nature of a fixed pain, situated over the outer side of the deltoid in the neighbourhood of emergence of the circumflex nerve. In the knee, it is usually referred to the inner condyle of the femur and tibia or to the under surface of the patella.

Apart from these localized pains, chiefly referred to and presumably dependent upon insidious changes in the intra-articular structures, there remain for our consideration certain paroxysmal pains of a neuralgic type. Beginning with those affecting the lower extremities, one may point out that it has long been recognized that sciatic pain may precede an outbreak of rheumatoid disease in the lower limbs. Spender thought that such sciatic pain might indifferently be followed by disease of the knee, hip, or tarsal articulations. I cannot confirm him as regards this sequence in the case of the hip and knee, inasmuch as in my experience it appears to be more especially related to disease of the ankle or of the tarsal articulations.

Here, again, I should like to draw attention to the fact that the sciatic pain which anticipates rheumatoid disease has certain special characteristics which I think are worth noting. In the first place, the pain does not radiate to all parts supplied by the nerve trunk, as it does so frequently in the classical sciatica, but is on the other hand usually confined to the thigh, running from the lower border of the buttock for a variable distance down the limb, sometimes so far as the upper limit of the popliteal space, but rarely if ever encroaching upon it. The usual painful spots associated with sciatica are not in evidence, but on the other hand there is often present "soreness" of the skin over the back of the thigh. Some describe the skin over the affected area as feeling "bruised," and pinching up the skin, even lightly, seems peculiarly objectionable.

From careful examination of a series of 100 cases, paying special attention to this point, I feel convinced that there is often present very real alteration in direction of hyperæsthesia and hyperalgesia, especially over the upper and outer portion of the posterior surface of the thigh, and in asymmetrical cases the contrast in this respect between the same areas in the unaffected and affected limbs is very striking. This pain, as I have said, seems especially related to early or oncoming disease in the tarsal or ankle joint, and coincidently the patients begin to complain of abnormal sensations in their feet, a common way of describing such being that the feet are the seat of a "burning" sensation, or that they feel at times "scalded." In others, again, coldness and numbness in the extremities attract attention, and in either condition local sweating of the soles is commonly a very troublesome feature. The muscles specially related to the articulation also participate in the abnormal sensations, and become the seat of more or less painful "cramps," affecting especially the plantar and the anterior tibial group, and the spasmodic contractions of the latter may jerk the toes upwards towards the knee. Walking for any length of time becomes increasingly difficult, and in cases where the disease begins in the mid-tarsal joint, the lack of elasticity in the patient's gait is very obvious. When walking on the flat, he as far as possible throws his weight on to the heel and the outer border of the foot. In going upstairs he avoids springing from the anterior portion of his sole, but lifting the foot up high he comes down upon his heel first and adopts the same plan in descending, seeking to avoid any transmission of pressure to the transverse tarsal joint.

Proceeding to an examination of the affected limb, we note that the plantar surface streams with perspiration, and this although the skin is unnaturally white or even bluish in colour, and to the touch feels cold and clammy. The contour of the foot is altered owing to more or less swelling over the dorsum and to the projection inwards of the head of the astragalus and navicular bone, while the instep is depressed and the sole appears flattened. If the general swelling be not too great, it may be possible to note the puffiness of the synovial sheaths of the extensors and of the peroneal tendons as they pass down behind the external malleolus. Not infrequently, the gluteal muscles on the same side may show fibrillary twitchings and atrophy.

The tendon reflexes also show important change, being in asymmetrical cases always increased in the affected limb, and the alteration in the superficial reflexes calls for special attention, inasmuch as two of them, the gluteal and the plantar, tend to exhibit the interesting phenomenon of associated action; thus, if the plantar reaction be sluggish, the gluteal reaction on the same side is likewise, and the reverse holds good. In male subjects, this variable though associated action of the gluteal and plantar reflexes was in sharp contrast to the cremasteric reflex, which is as a rule exaggerated on the affected side, and the difference in the response of all three reflexes to those elicited in the opposite and unaffected limb was very striking in asymmetrical cases.

The correlation of this group of symptoms with early or oncoming rheumatoid disease of the ankle or tarsal joints is, it seems to me, worthy of more than passing notice, and I would just note the following points :—

Head, whose work on spinal distribution has done so much to unravel the hidden secrets of neurology, has shown us that the plantar and gluteal reflexes own allegiance to the same segments of the spinal cord, and when we bear in mind that those muscles which play a prominent part in the maintenance of the plantar arch, i.e., the peronei, anterior tibial muscles, and intrinsic muscles of the foot, are governed by the same segments (first and second sacral), we seem to gather fresh light as to the possible etiology of the flat-foot so common in rheumatoid patients.

Moreover, these same segments are responsible for the nutrition of the gluteal muscles, the sensory innervation of the skin over the sole of the foot, the back of the thigh, and the popliteal region, and they also in part control the secretion of the sweat glands of the foot. In an article communicated to the *Lancet* in 1902, I drew attention to this tendency to a segmental distribution on the part of these various phenomena in some cases of rheumatoid arthritis, the observation being based upon an examination of one hundred cases of the fusiform type (atrophic of Goldthwait), and it is interesting to record that McCrae, in his bulletin on the subject, states that "in addition to the deep reflexes, the superficial ones are often much exaggerated, which we have found especially true of the cremasteric reflex," and in addition, he noted "the curious association of alteration in reflexes, such as between the gluteal and plantar, etc," suggestive, as he states, of a segmental distribution.

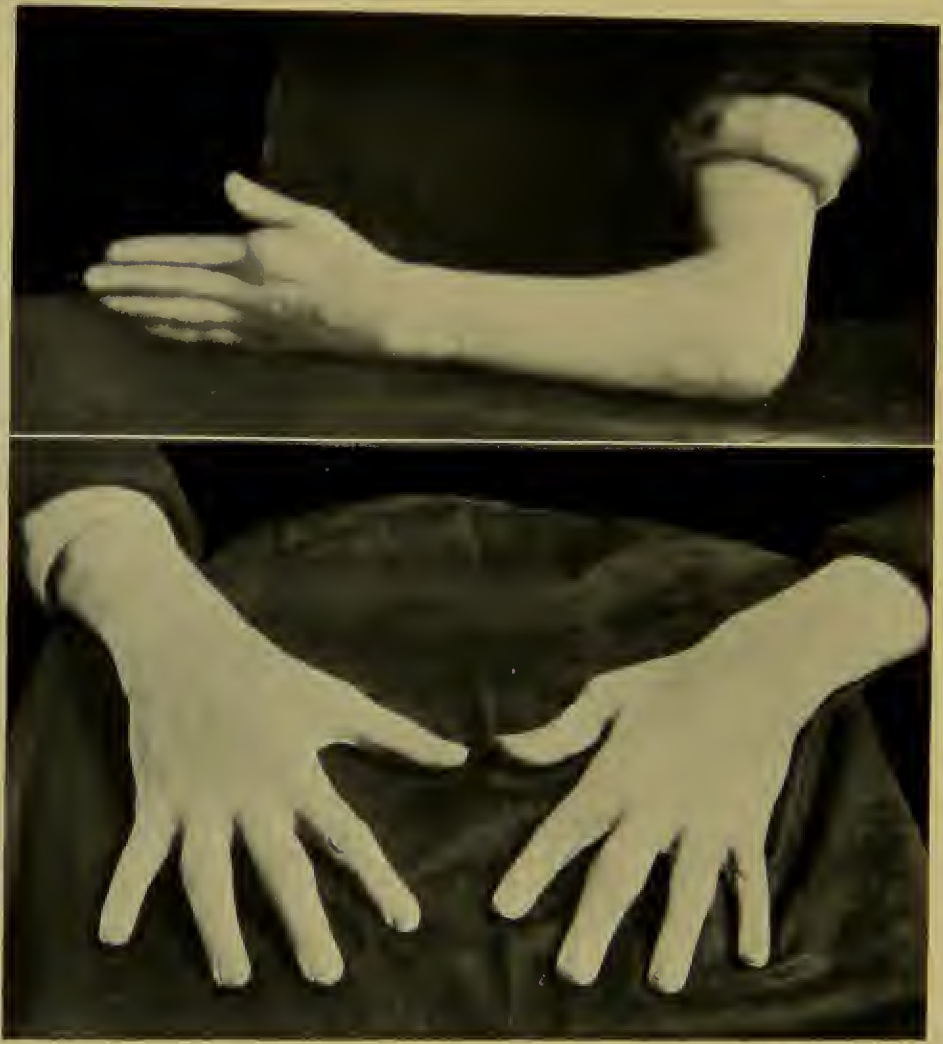
Similarly in the upper limb the oncoming of rheumatoid disease may be heralded by pains, which, like those of the lower limbs, may be either lancinating or dull and aching in character. Spender noted that such pains might run along the radial border of the forearm, following the course of the musculo-cutaneous nerve.

Personally I can confirm him in that pain is often present along the radial or pre-axial border of the forearm, both anteriorly and posteriorly, but in my experience such pain does not confine itself to the territory of the musculo-cutaneous nerve, and stop short at the wrist, but on the other hand it extends into the thumb and the index finger, thus trespassing upon the area supplied by the median anteriorly and the radial posteriorly. This area is one of the commonest sites of pain in rheumatoid arthritis. It is often attended by marked superficial "soreness," or it may be described as feeling "bruised." Objectively tested, the area shows fleeting hyperæsthesia and hyperalgesia, followed by dulling of sensation, all of which sensory changes in these early stages are fleeting. In rare cases the overlying skin of this area may show signs of œdema, and frequently the pigmented spots noted by Spender make their appearance insidiously. Incidentally it will be remembered that Spender failed to identify the distribution of these spots with any nerve track. Not infrequently, sharp lancinating or "stinging" pain over this pre-axial area initiates an attack of local syncope and asphyxia in the index and thumb. In some cases also this pain is accompanied by a "stiffness" of all the fingers, disabling the patient from bending them except by pressure, and presumably due to extensor spasm. Finally, this pain often precedes enlargement of the joints of the finger and thumb.

Fusiform enlargement of the ring and middle finger is not uncommonly preceded by pain, which may be very severe. In their case the main track of the pain is experienced along the mid-dorsal surface of the forearm and hand, radiating into these digits.

In some cases this dorsal pain is accompanied by pain along the mid-ventral area of the forearm, and hand and flexor spasm of all the fingers. Hyperæsthesia and hyperalgesia, followed by numbness over the painful area on the dorsum, are present during the paroxysms. I have also seen localized sweatings over this mid-dorsal area of the forearm. In addition to the two above tracts of pain related to oncoming disease in the joints of the index and thumb and ring and middle fingers respectively, there remains the occurrence of pain running along the ulnar or post-axial border of forearm and hand into the little finger, which may precede swelling of the wrist and the

PLATE III.



Upper Figure:—Shows early and coincident involvement of the metacarpo-phalangeal joint of index finger and the superior radio-ulnar articulation.

Lower Figure:—Symmetrical disease of the mid-phalangeal articulations of the ring and middle fingers (segmental arrangement).

joints of the little finger. The same fleeting sensory alterations accompany this pain as have been described in connection with the other two painful tracts.

As regards the relationship of this ulnar pain to the disease in the wrist joint, it seems to me to be especially related to disease of the radio-carpal portion of the articulation, but the radio-ulnar portion of the articulation seems on the other hand to be related to the pain previously described which runs along the radial or pre-axial border of the forearm into the index and thumb. I have come to this conclusion because I have noted in cases of the index and thumb type the superior and inferior radio-ulnar articulations are often simultaneously attacked, and supination of the forearm is early limited in range, and this limitation may occur long before flexion of wrist or elbow joints becomes either painful or restricted. On the other hand, in cases where pain along the ulnar or post-axial border of hand has been in evidence, flexion of the wrist joint has been seriously limited and in some cases abolished. It will be noted that I have described three tracts of pain in the forearm and hand: (1) the pain along the radial or pre-axial border of the forearm, related to disease in the index and thumb, and also radio-ulnar articulations, (2) pain along the mid-dorsal surface of forearm, associated with disease in the ring and middle fingers, and lastly, (3) pain along the ulnar side of forearm and hand, related to disease in the wrist and little finger and radiocarpal joint.

In endeavouring to account for these various phenomena correlated with early rheumatoid disease of the joints of the index and thumb on the one hand and similar changes in the ring and middle fingers on the other, I would point out, first, that this grouping of the joint lesions is by no means uncommon, and in this observation as to their segmental distribution I am confirmed by McCrae, who states as follows: "The association of the reflexes often follows their segmental distribution, as may also the curious localized distribution of the lesions in the hands. Thus the middle and ring finger only may be involved or show special changes. In another case the thumb and first finger may show the same association."

In concluding this attempt to describe the premonitory symptoms met with in what Teissier has recently termed "the period of invasion" of rheumatoid arthritis, I would lay stress on the following points :—

1. That these so-called "dead" blue or white fingers, muscular cramps and sensory changes are very frequent forerunners of rheumatoid arthritis.

2. That their appearance in persons recently the subjects of some infectious disorder, influenza, etc., points to an imperfect convalescence, and indicates that every attempt should be made to ascertain the source of this tardy recuperation, and that such, if found, should be promptly treated.

The same inference of toxic absorption is to be drawn from their occurrence in women recently confined, or recovering from the effects of a miscarriage, or depressed by prolonged lactation, and in such cases the possibility of some overlooked condition of uterine sepsis should be borne in mind, and if present corrected.

Again, in any case where the patient gives a history of some previous polyarthritic attack supposed to be "rheumatic fever," the presence of the above signs should immediately suggest a revision of the diagnosis in favour of the past joint disorder being rheumatoid rather than rheumatic in nature.

The same remarks as to the significance of these phenomena apply to their appearance in connexion with any gastro-intestinal affections, namely, that they denote some degree of toxic absorption; and this view of their nature carries with it also the indication that all localized foci of absorption should be treated, if present, and failing their detection, the *vis resistantiæ* of the individual should be strengthened by all means in our power.

REMARKS ON PREMONITORY SYMPTOMS.

In reviewing collectively these groups of premonitory symptoms, it may be justly argued that there is nothing distinctive about the various phenomena included under this heading. They are certainly very commonly to be met with, and occur moreover very often in patients who never develop arthritis. For instance, some of them, more especially the sensory, are very similar to the early symptoms met with in the various forms of multiple neuritis. Others, again—for example the vasomotor phenomena—are apparently clinically identical with those of Raynaud's disease, while the muscular cramps, as we shall show later, have many points in common with tetany.

It can hardly be doubted that in all the various diseases in which these symptoms may appear, the underlying pathological condition is one of toxæmia. If we go still further afield in these chronic toxæmias we note still the same overlapping of symptoms. Tingling and numbness, local syncopes, are but small clouds on the clinical horizon, but they may be the sole tokens of commencing cord degeneration in pernicious and other anæmias, as recent research has shown. Tingling and numbness of one hand were the only symptoms complained of by a lady who recently consulted me. Otherwise, beyond being slightly anæmic, she was to all appearances perfectly well, but fortunately a visit to her dentist revealed the fact that she was suffering from pyorrhœa alveolaris, and treatment directed to this condition of oral sepsis was followed by disappearance of her unpleasant symptoms, and for aught we know this timely intervention may have prevented the onset of severe, possibly pernicious, anæmia. Similarly neuralgic pains in the limbs may mean a foul stomach, gout, rheumatism, or what not, but it is at any rate prudent to remember that they may be the clinical outposts, so to speak, of diabetes or commencing tabes, lest haply a sudden coma or an equally sudden spontaneous fracture startles us into remembering that symptoms are but indications, not diseases, and are to be regarded, as the sagacious and far-seeing Laycock once said, "only as minute in a popular sense." The pathway of the physician is strewn with such pitfalls, and the lesson is obvious to "despise not the day of small things," and I would suggest that while we remember that such slight symptoms may be the only signs of insidious and mortal diseases, we should not forget that they may be the forerunners of rheumatoid arthritis, which, though it does not kill, may yet chain a man for years, like Heine, to a "mattress grave," until some chance shaft from the quiver of Azrael puts an end to his long-extended sufferings.

These illustrations, drawn from the various chronic toxæmias other than rheumatoid arthritis, show the impossibility of claiming any specific relationship between these symptoms and rheumatoid arthritis; neither am I desirous to convey any inference other than that they are signs of chronic toxæmia, and as such worthy of more recognition than they have hitherto received. Whether rheumatoid arthritis be due to an infection by one or many germs, or be of the nature of auto-intoxication, presumably in any case the morbific influence is exerted through the medium of toxins, in other words, it is due to a toxæmia, one moreover in the majority of cases essentially chronic, slow of evolution, cumulative in its consequences. Is it likely, having regard to the slow, insidious progress of many cases of this disease, that the first symptom of the chronic toxæmia should be a fully developed peri-articular swelling? Is it not infinitely more probable that in the presence of such a general toxæmia the more delicate and sensitive nervous structures should respond more swiftly than the peri-articular or synovial structures? Have we not here the clue to the nature of the vasomotor phenomena, the cramps and so forth, namely, that they are the outward expression of a cord whose reflex excitability is heightened as the result of toxic invasion. We are, I am sure, too prone to look upon the joint swellings when they appear as a new departure, as a something which has descended upon the unhappy patient like "a bolt from the blue," and disregarding these premonitory symptoms, to lose our link with the past and join the cloud of witnesses who testify to the cryptic origin of so many cases of rheumatoid arthritis. My own conception of the disease is that it starts, so to speak, from a pathological groundwork of toxæmia, of which the primary manifestations are these said premonitory symptoms, which in its presence proceed to display themselves.

At this stage on one, unless endowed with prophetic instinct, could foretell their future lines of development. In cases where the toxæmia is presumably transient, they may stop short and no more be heard of them, or, on the other hand, peri-articular swellings may follow in their wake, and straightway we are confronted with the clinical picture of rheumatoid arthritis.

Let us take an example. Suppose a patient, a woman, depressed and debilitated it may be by a severe confinement or chronic dyspepsia, comes under our notice complaining of cramps in her hands. A provisional diagnosis of tetany is made, having regard to the frequency with which these symptoms appear in nursing women and dyspeptics. Supposing, however, she does not consult us until a later period, when she comes before us complaining of her finger-joints, which on examination are seen to be the seat of spindle enlargement. Her attention being naturally centred on these objective signs, she perhaps says little or nothing about her previous illness with its associated cramps, and all too frequently we likewise are only too apt to consider we have done all when we have christened the affection rheumatoid arthritis, thus overlooking the true sequence of events—prolonged lactation or dyspepsia, muscular cramps, peri-articular swellings.

Here it seems apposite to refer to the fact that apart altogether from the question of rheumatoid arthritis, peri-articular swellings have been known to supervene in tetany. Now our forefathers termed such cases examples of "rheumatic tetany."

Their supposed rheumatic origin is now exploded, but apart from this error, the term had this advantage, that it clearly carried with it the inference that the joint swellings were but a further elaboration of the original pathological process that gave rise to the antecedent cramps. Similarly, in Raynaud's disease, apart from any relationship to rheumatoid arthritis, joint swellings have been known to supervene in sequence to these phenomena.

Consideration of these and other analogies has led me to conclude that the muscular cramps and other premonitory symptoms are but the primary manifestations of the chronic toxæmia, which, cumulative in its consequences, culminates in the clinical condition we term rheumatoid arthritis.

In reply to those who doubt the existence or dismiss as trivial these signs of oncoming rheumatoid arthritis, I would remind such of the old classical saying, "*Nihil natura agit frustra.*"

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Chapter VIII.

CLINICAL ACCOUNT.

In the preceding chapter we were chiefly concerned with the symptoms peculiar to what Teissier terms "the period of invasion" of rheumatoid arthritis, namely, those which precede the development of joint swellings.

We pass now to consider the features of the mature disease, dealing first with its modes of onset. These are very diverse; so variable, indeed, that any classification of them can only be approximate. With this qualification, however, they may be divided into acute, subacute, and chronic.

In the first-named—*acute*—the disease in rare instances may commence quite abruptly (*Plate IV*).

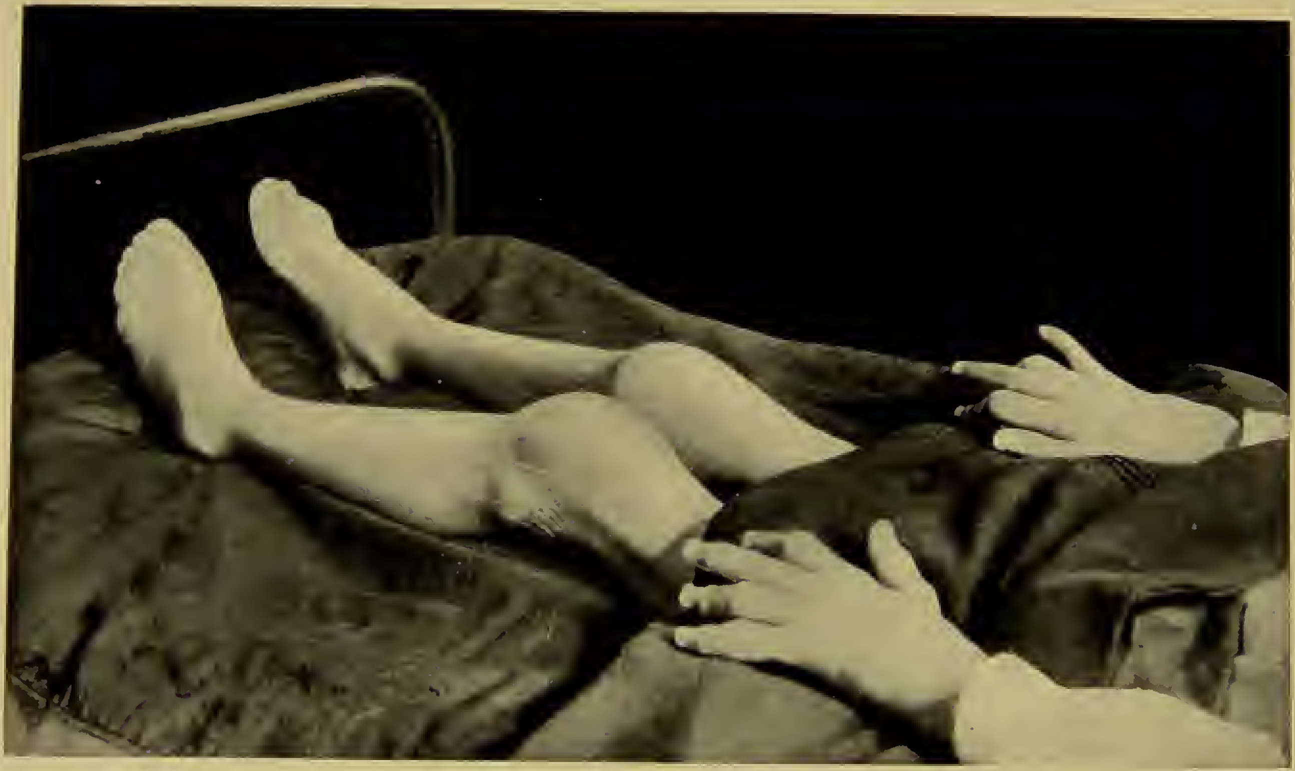
Without any previous malaise a rise in temperature takes place, with coincident enlargement of one or more of the smaller joints of the hand, the disease, in its further course, spreading with merciless rapidity from joint to joint.

It is in these cases characterized by swift generalization of the joint swellings that confusion with acute rheumatism is most likely to occur.

In some examples, however, of this acute type, this sudden incidence of joint swelling with pyrexia has been preceded by an indefinite period of lowered health, frequently traceable to an antecedent illness. During this period of diminished resistance, I have often been able, from a careful examination of the previous history, to satisfy myself that the patient had experienced some of the premonitory symptoms before alluded to. The subjects of these acute forms are almost invariably young females.

In the *subacute forms* the initial rise of temperature is more moderate, the pyrexia less continuous, and the progressive involvement of the joints more gradual. Indeed, the course pursued in this variety can best be described as a series of comparatively brief attacks of polyarthritis with pyrexia; from

PLATE IV.



Acute Rheumatoid Arthritis, involving nearly all joints of both upper and lower extremities.

the earlier of which the joints may, in some cases, apparently recover.

On the other hand, with the later attacks the impairment of the joints becomes more and more marked, and the peri-articular swellings tend to persist, till eventually we arrive at the typical picture of a generalized rheumatoid arthritis.

During the apyrexial periods that intervene, the muscular cramps and vasomotor phenomena mentioned previously persist, linking up the series of attacks into one continuous chain of events.

Finally, in the *chronic forms* the disease arises in a most insidious fashion, the implication of the joints taking place very slowly but progressively, while the temperature remains normal throughout.

Depression of spirits, with fretfulness and irritability of temper, are usually met with in these cases, in addition to the other prodromal phenomena I have described. This last mode of onset is most frequently met with in those cases which arise at or near the menopause.

ARTHRITIC PHENOMENA.

The Arrangement of Joint Lesions.—The joint swellings constitute the salient and most conspicuous feature of the affection around which the collateral phenomena group themselves.

The disease evinces a striking predilection for the smaller joints of the hands, its earliest manifestations being usually localized in the metacarpo-phalangeal or mid-phalangeal joints of the fingers.

The terminal phalangeal articulations may in very rare instances undergo enlargement. More commonly they become fixed in some abnormal position without enlargement.

If examined closely, however, they will be often found to exhibit signs of having undergone atrophy of the articular structures and overlying skin; thus contrasting with the mid-phalangeal joints, in which the ultimate atrophy is preceded by a stage of fusiform swelling of these articulations.

As before stated, there is a tendency to segmental arrangement of the joint lesions in the fingers. In some instances the mid-phalangeal joints of the ring and middle fingers will alone be implicated, and this symmetrically, while in other cases the swellings may be limited to the joints of the thumb and index finger in both hands.

The disease, as Charcot[1] long since remarked, tends to spread centripetally up the limbs; and in accordance with this the joints of the wrist and elbow are involved, either simultaneously, or in quick sequence to the joints of the hand.

While in the vast majority of cases the disease commences in the joints of the fingers, it may, on the other hand, begin in those of the toes, either the mid-phalangeal or the metatarso-phalangeal articulations. Subsequently, the tarsal articulations, the ankle, and knee, are implicated.

The hip and shoulder joints, in the opinion of most authorities, enjoy immunity; but, according to Painter,[2] this exemption is more apparent than real, the involvement of these joints, when it occurs, being overlooked, owing to their less exposed situation.

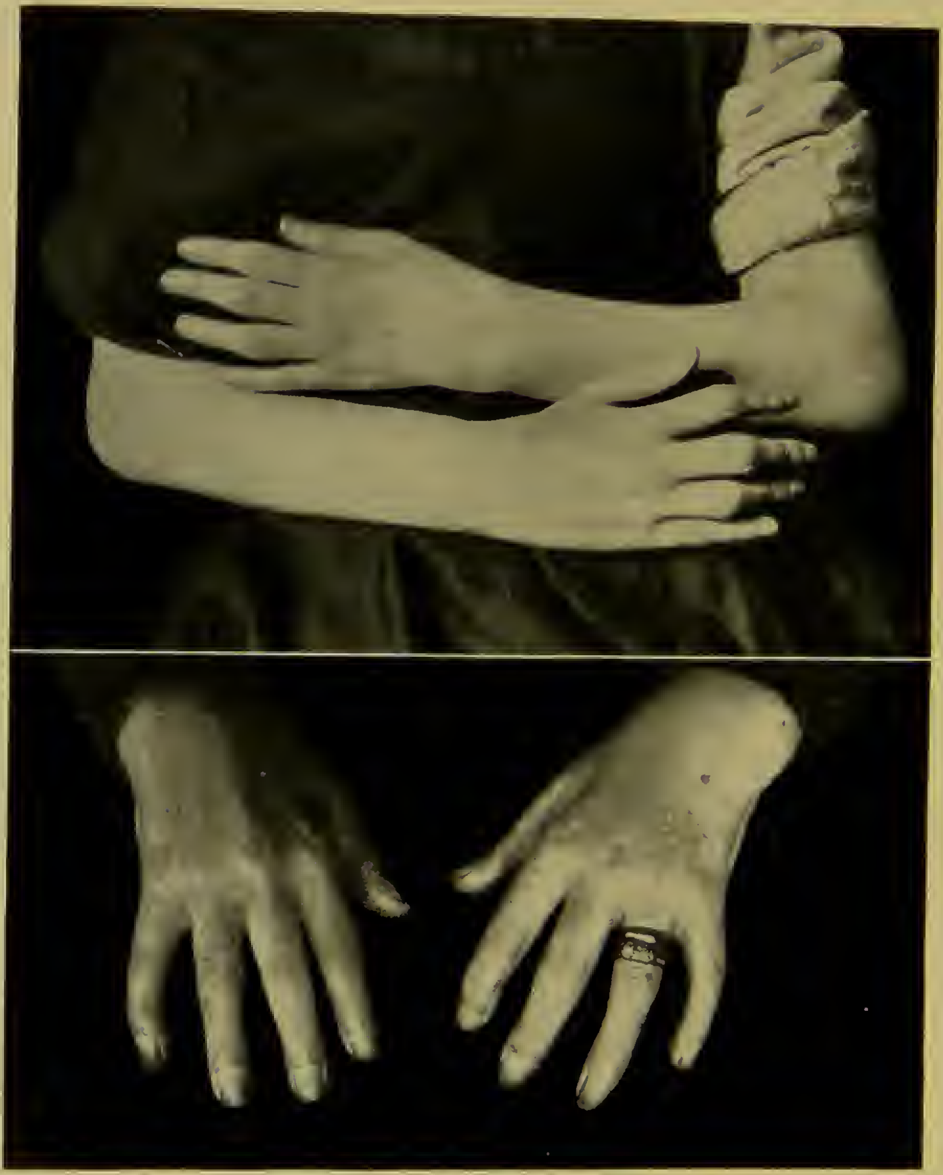
The centripetal spread of the lesions, though undoubtedly very typical of rheumatoid arthritis, is at times departed from, the larger joints being affected before the smaller, the disease travelling down the limb, so to speak, from the elbow or knee to the hands or feet.

The joints of the hands and feet may be simultaneously implicated. One peculiarity of the distribution of the lesions remains to be noted, namely, the pathognomonic frequency with which the temporo-maxillary joints are attacked, and, in addition, those of the cervical spine. Not infrequently slight involvement of the jaw and vertebral articulations may be overlooked, the patient's almost ubiquitous suffering diverting his attention from these joints, in which the morbid process does not, as a rule, pursue an inveterate course in these more generalized cases.

Occasionally extensive affection of the spinal column is present in rheumatoid arthritis; but I have thought it best to defer more detailed description of this special feature to the section dealing with spondylitis deformans.

According to Havilland Hall[3] and Casselberry,[4] the arytænoid joints may be involved, while according to other observers the joints in the middle ear in some instances do not escape the

PLATE V.



Upper Figure:—EARLY STAGE. Showing fusiform enlargement of mid-phalangeal joints.

Lower Figure:—LATER STAGE. The swelling of joints has disappeared, the fingers are fixed in extension, with marked atrophy of the overlying skin, and nearly complete obliteration of wrinkles, especially over mid-phalangeal articulations of ring and middle fingers.

ravages of the disease. One of the most remarkable features of the affection is the symmetry with which the articular lesions are distributed. As Sir Thomas Watson,[5] in his lectures, states, "To use a paradoxical expression, the deformity is symmetrical; one crooked joint is just the copy of his fellow." Of the substantial truth of this observation, first made by Dr. William Budd[6] in 1842, anyone can convince himself. The symmetry also extends to any abnormalities of the skin lying over the articulation, whether pigmentary or atrophic in nature; and, according to Garrod,[7] this remarkable likeness extends itself even to the deeper structures of the articulation, the cartilages betraying the same symmetry in the lesions affecting their substance. It must be remembered that it is only in the swiftly progressive forms that this condition of symmetry is rapidly arrived at, corresponding joints on opposite sides being in point of time coincidently involved. In the majority of cases at first the condition is asymmetrical, although ultimately by the spread of disease the same striking symmetry is arrived at.

In the more slowly progressive forms the articular lesions are seen in various stages of development, some showing the earlier condition of spindle enlargement, while others, more advanced, have undergone the contraction or shrinkage that ensues during the later periods of the affection.

Clinical Characters of Joint Swellings.—The external appearances are best studied in those joints which, by reason of their exposed situation, more readily admit of examination, such as those of the proximal phalangeal articulations (*Plate V*).

The joint is the seat of fusiform, or spindle-shaped, enlargement, the skin over which is waxy white or semi-asphyxial in tint. Not infrequently the swelling shows a slight constriction at the line of the articulation.

On palpation the swelling may feel remarkably tense and elastic, and in some instances apparently fluctuates. In others, however, the enlargement is softer and less resistant, giving more the impression of thickening in the synovial capsule; but in no case at this stage can we detect nodular outgrowths at the line of the articulation as in osteo-arthritis. Despite the suggestion of fluctuation conveyed to the fingers, there is rarely any great excess of fluid in the joint.

This spindle-shaped enlargement, though more conspicuous in the above joints, is not confined to them, for the same outline is easily distinguished in the knee and elbow. Similarly, in the wrist, the peri-articular thickening is easily defined, giving rise to a domelike swelling with ill-defined borders on the dorsum of the articulation, shading gradually on its proximal side into the forearm with its wasted muscles, while the swelling at its distal side contrasts with the neighbouring sunken interosseous spaces.

In the knee the capsule of the joint is clearly outlined by swelling, due partly to effusion, partly to the thickening of the synovial layer of the capsule. The effusion in isolated cases may be excessive, and in very rare instances follows the peculiar course of an intermittent hydrarthrosis, while the bursæ communicating with the joint may participate in its unusual distention.

Similar appearances are to be met with in the ankle joint; and in this case the extensor tendons on the dorsum may show effusion into the sheath, also the peronei, giving rise to an elongated swelling extending down behind the external malleoli.

It must not be forgotten that where the joints are the seat of extensive villous formations all the symptoms of intra-articular effusion may thereby be simulated. This leads us on to note the clinical symptoms of villous hypertrophy, that condition so commonly engrafted upon rheumatoid arthritis. It occurs also in osteo-arthritis, but much more rarely. The hypertrophy of the synovial fringes in the former is generalized over the whole extent of the synovial membrane, and not restricted to any particular area. In osteo-arthritis, on the other hand, according to Painter and Erving,[8] as ascertained by operation, though it may be generalized, it is often localized, and due to internal traumatism, the result of irritation of the synovial membrane by projecting bony spurs.

Although this villous overgrowth may be widely generalized in the joints, as a rule it is only in the knees that symptoms indicative of its presence arise. The villous condition of the synovial membrane being uniformly distributed, there is general enlargement of the joint, whereas in localized forms of the condition due to trauma the enlargement is more limited, being either restricted to the region above the level of the patella, or limited to one below it. On passive movement of the joint a creaking noise may be elicited, owing to the friction between opposed fringes. The fringes reaching a certain size act as foreign bodies, and are a source of continual irritation in the joint, giving rise to pain and restriction of movement, with a sensation of catching, due, presumably, to their intrusion between the articular surfaces. Some degree of effusion may follow; but never so marked as occurs in purely traumatic forms. Occasionally their presence may be evident to palpation as soft, slippery bodies, of a doughy nature, not unlike a bag of worms. The most likely sites for them to be detected are the inner side of the ligamentum patellæ, or the pouches lying over the femoral condyles, and in the ankle along the anterior surface of the joint below and in front of the malleoli.

COLLATERAL PHENOMENA.

Inasmuch as the joint lesions constitute, so to speak, the central feature of the disease, I have adopted the term "collateral" to designate those additional phenomena of the malady referable to disturbance of the muscular, vasomotor, and sensory systems. These latter are in reality an extension of those previously treated of under the title of "Premonitory Symptoms." But, inasmuch as these continue to manifest themselves subsequently to the appearance of joint swellings, the term "premonitory" ceases at this stage to be applicable to them. At one time prodromal symptoms, they have now become collateral features, their presence bestowing on the disease its distinctive facies as opposed to all other joint affections.

Muscular System.—Undue irritability of the muscles, as we have shown, appears to be one of the early symptoms of oncoming rheumatoid arthritis. I have already entered into a detailed description of these cramps "or spasms" as they occur before enlargement of the joints supervenes. It now only remains for me to note their persistence after peri-articular swellings have appeared; and, in addition, their somewhat close similarity to the muscular spasms of tetany, as evidenced by the extreme faithfulness with which the forced attitudes typical of that affection are reproduced in rheumatoid arthritis, such as the conical or "accoucheur's" hand, and the various degrees of the "main en griffe." These strained positions are obviously muscular in origin, inasmuch as they undergo gradual correction with the cessation of spasm, and allow of rectification during the spasm by passive movement.

It seems to me, therefore, that the conditions of ulnar deflexion of the hands and fingers, likewise the radial deflexion (*Plate VI*) more rarely met with, are in the early stage due to muscular spasm; and the fixity of these malpositions at a later date is, I think, due to the subsequent atrophy and contracture of the related muscles. Again, the clinical similitude of these spasms to those of tetany is further enhanced by the fact that it is possible to evoke Chvostek's symptom (percussion of the various nerve trunks giving rise to marked contraction of the particular muscles innervated by them).

I have often been able to evoke such by percussion of the ulnar, median, facial, and sciatic nerves. Furthermore, this increased mechanical excitability of the nerve trunks in the early stages of rheumatoid arthritis is, I believe, in some obscure way related to the joints affected, inasmuch as I have, in asymmetrical cases of the affection, been able to elicit the phenomenon in the affected limb, and not in the opposite member in which joint swellings were not present. It is certainly lost after a time, and is, I think, confined to the early stages of the disease.

Again, the electrical reactions in some instances approximate to those of tetany; response to faradism was sluggish, but brisk to galvanism. A.C.C. response was often equal to K.C.C. In one instance, in association with Mr. J. R. Benson, we obtained that peculiar reaction distinctive of tetany, anodal-opening tetanus. Diamantberger[9] has also been struck by this resemblance to tetany, and cites an instance in which this diagnosis had been arrived at before periarticular swellings appeared to establish the true nature of the affection.

Ballet and other authorities have also recorded cases in which muscular spasms lasting for hours were prominent features in individual instances.

Muscular Tremor.—Disorders of co-ordination, such as tremor, are sometimes present in rheumatoid arthritis. Such may be localized in the asymmetrical cases to the affected limb; or in cases with symmetrical joint involvement the tremor may be generalized; but to this form I shall refer more in detail in a later section.

The athetotic movements of the hands, thought by Spender[10] to be not infrequent, are in my experience extremely rare.

Muscular Atrophy.—This constitutes one of the most conspicuous features of the malady.

In the initial stages the selected affinity of the disease for certain muscles can be observed.

Thus the extensor group suffer more than the flexor; while the interossei are amongst the earliest muscles to show diminution in their size.

The inner two dorsal interossei are often early picked out; but, according to Gowers,[11] the abductor indicis suffers more severely.

The thenar eminences also may become much flattened.

In advanced cases, however, the flexor groups may be found quite as wasted as the extensor muscles.

This muscular atrophy once established is extremely persistent.

It may be not only intense in degree, but widespread in its distribution, and when such is the case it imparts to the disease a remarkable resemblance to some affection of the central nervous system.

As Osler says, "There are cases of rapid muscular wasting, symmetrical involvement of the joints, increased reflexes, and trophic changes, which strongly suggest a central origin."

Now this swiftly induced and severe muscular atrophy, if not actually independent of, is not inevitably associated with, unusual severity of joint lesions.

On the contrary, in cases in which the arthritic lesions are severe, the degree of muscular wasting may be comparatively slight; while, on the other hand, examples with slight joint changes may show intense muscular atrophy.

Again, the problem is complicated by the fact that cases of approximately the same duration, and, as far as can be judged, with a similar degree of joint trouble, may manifest very unequal degrees of atrophy.

Such discrepancies between the arthritic lesions and the degree of muscular atrophy produced, when taken together with the occasional wasting of muscles remote from the affected articulation, are difficult to explain on the basis of a reflex arthritic muscular atrophy.

I note that the late Douglas Kerr,[12] of Bath, dealing with this question of muscular atrophy and contracture in rheumatoid arthritis, states, " that joints quite free from the rheumatoidal attack are frequently affected by these muscular contractures, and may become fixed, or terribly distorted and dislocated." The inference presumably to be drawn from this statement is that the muscular atrophy and the joint lesions are not inevitably correlated, and may occur independently of each other.

Such considerations as these led Ord to compare the disease from this point of view with progressive muscular atrophy. Personally, I am inclined to think that it presents, at any rate in the most generalized forms, a striking resemblance clinically to amyotrophic lateral sclerosis—and this, by reason of the fact that in certain cases rapid wasting of apparently all the muscles in the body ensues.

Not only do the muscles of the extremities atrophy, but also those of the thoracic and abdominal groups ; while those of the face enjoy no immunity. At the same time, marked exaggeration of the deep reflexes takes place, which enhances the suggested similarity to the above affection.

The occasional marked implication of the facial muscles is shown by the following notes of a case under my own observation. The disease was of five years' duration, and there were present the following signs : Left ptosis, marked limitation of movement of internal and external recti in the same eye, inability to keep the left eye closed against resistance, extreme sluggishness of pupil, notable paresis of left facial muscles, including orbicularis oris, atrophy of masseter and temporal muscles, with fixed anæsthesia over the left trigeminal area.

Through the kindness of Preston King I have recently had an opportunity of examining a case under his care which presented similar signs of cranial nerve involvement.

The subject, a woman 40 years of age, was attacked by rheumatoid arthritis some six months ago. Coincidently, signs pointing to paralysis of the right third nerve presented themselves—a widely dilated pupil, slight ptosis, and diplopia. The facial muscles on the same side showed distinct paresis.

Spender, as we know, cited instances which he considered pointed to an extension of the disease upwards giving rise to symptoms suggestive of some degree of bulbar palsy.

In two examples which came under his notice he observed a growing feebleness in the cervical and intercostal muscles. With this were associated feebleness of the pterygoid and masseter muscles, inability to protrude the tongue to the usual extent, hesitancy of speech, while the face took a blank or masklike expression, especially in its lower half. In addition, cyanosis, with short and panting respiration, was present.

The following cases appear worthy of reference in this connexion:—

Oliver,[13] in a discussion on "arthritis deformans" at Newcastle-on-Tyne, refers to the association of bulbar palsy with this disease, quoting a case in which the arthritic changes supervened secondarily to the bulbar palsy.

In a recent discussion at the Bournemouth Medical Society on this same disease Dr. Fred. Gardner quoted an example, the following notes of which he has kindly furnished:—

The patient, a lady suffering from rheumatoid disease for forty years, had been under his care for the last ten years. Trophic disturbances were very marked in her case, ulceration appearing in parts not especially exposed to friction. Latterly the tongue has become swollen and the lips slightly paralyzed, so that she talks thickly, saliva dribbles from the mouth, and when speaking there is a fine tremor affecting the buccinators. Speech is slow, and words run into one another. In addition there has been difficulty of swallowing at times, generally referred to "soreness of throat," although there are no signs of local inflammatory trouble.

Before concluding our remarks on muscular atrophy it should be mentioned that in very rare instances the wasted muscles may give the reaction of degeneration. I have met with one instance myself in a young female, whose case presented the following features: She was the subject of widespread rheumatoid arthritis. She had completely lost the power of extending her left leg upon the thigh, though passive extension of the knee was both easy and painless. The extensor muscles of the thigh on this side were extremely wasted, and the knee-jerk apparently abolished. The quadriceps extensor on electrical testing gave complete reaction of degeneration. The contraction of the adductors and hamstrings was sluggish, reacting only to currents of painful intensity.

Myotatic Irritability.—Passing now to the consideration of the reflex reactions in rheumatoid arthritis, the study of these in asymmetrical cases soon convinces us that the deep reflexes are much more brisk on the diseased side than on the other, and this holds good not only of the lower extremity, but also the upper, the wrist and triceps jerks being increased to the same extent as the knee and Achilles jerks in the lower limb. The knee-jerk may show a remarkable degree of exaggeration, and knee-clonus may also occasionally be elicited. Ankle-clonus may be evoked in some instances, and its presence in one limb, while absent in its fellow, often constitutes a striking phenomenon in asymmetrical cases. The same high grade may be reached in reflexes of the upper limb, while, as before stated, the jaw-jerk, when that articulation is diseased, may readily be forthcoming.

Finally, no remarks on the tendon reflexes are complete unless we mention the fact that they are occasionally extinguished. Similarly, the superficial reflexes in the asymmetrical cases—gluteal, plantar, and cremasteric—are either exaggerated or sluggish, the two former, as I have before stated, displaying striking harmony in their reactions. If the plantar be sluggish the gluteal reaction on the same side is likewise, and the reverse holds good, while the opposite and unaffected limb gives normal reactions. Exaggeration of the plantar reflex when present may reach a high grade, and in some cases shows a tendency to spread to the opposite limb, whose tensor vaginæ femoris will be seen to contract vigorously; but in such cases I was not able to evoke a bilateral reflex by stimulating the sole of the opposite unaffected limb. I have in solitary instances seen a definite extensor response to plantar stimulation.

We have hitherto confined our attention to the reflexes in asymmetrical cases, and it only remains to add that when both limbs are affected, or possibly all four, the deep reflexes in all cases will be exaggerated. The increase in myotatic irritability is one of the earliest features to appear in the clinical picture. It is one of the last to fade away. It maintains its high grade long after the joint troubles are seemingly quiescent, and I am sure that any unusual persistence at a high level is significant of an imperfect convalescence, and the possibility of a fresh outbreak is not remote.

On the other hand, diminution is of good omen. In the visceral reflex mechanisms one rarely finds any appreciable change.

I may mention that in two children, aged 7 and 9 respectively, with rheumatoid arthritis, there were present in both incontinence of urine, and in one incontinence of fæces. These symptoms came on since the onset of the disease.

VASOMOTOR PHENOMENA.

These manifestations, so frequently met with in what may be termed the period of invasion in rheumatoid arthritis, form also some of its most interesting collateral features. Like the muscular atrophies, they may vary much as regards their degree of development in individual instances ; they may be extremely severe and widespread, involving the genitals, as well as the hands, feet, ears, and nose, examples of which I published in the *Edinburgh Medical Journal* some years ago. On the other hand, they may be slight or altogether absent. They are not necessarily associated, even in their extreme types, with unusually severe arthritic lesions. I have often noticed their prominence in cases in which the arthritic lesions and muscular atrophy were not marked.

There is no necessity to recapitulate in detail the individual features of the paroxysm, inasmuch as they are exactly similar in all respects to those of the premonitory period, being, in fact, as far as one can ascertain, merely a continued expression of them. This much, however, may be said, that in those cases of rheumatoid arthritis which pursue an intermittent course, namely, one characterized by a series of acute attacks, the vasomotor phenomena by the increase in their severity will foreshadow the oncoming attack, while their gradual wane and possible disappearance is of good omen from a prognostic point of view.

Their similarity to Raynaud's disease, which I have before pointed out, is enhanced by the fact that some of the rarer manifestations of that disorder are to be found in those cases of rheumatoid arthritis distinguished by the presence of these vasomotor phenomena. Raynaud noticed that either coincidently or alternatively with asphyxial conditions in the extremities transient obscurations of vision might occur. He was confirmed in this statement by Galezowski, who further noted temporary alterations in the fundus oculi, the retinal arteries evidently undergoing spasm, attended here and there by partial constriction of their calibre, being definitely narrower round the papilla than at the periphery of the retina.

Rheumatoid patients suffering from local syncope or asphyxia often complain of transient "mistiness" or "fogginess" of vision; and, therefore, in company with Mr. Beaumont, of the Bath Eye Infirmary, I took the fields of vision of fifty patients showing these vasomotor features. The fields in all were uniformly contracted. Such retraction was not obtained in cases when the peripheral vasomotor disturbances were not marked. In order to confirm the vasomotor origin of these troubles the visual fields were taken before and after the inhalation of nitrite of amyl. Expansion of a transitory nature followed, but in no case did it reach the normal periphery. It seems, therefore, reasonable to conclude that as a result of the constant recurrence of the periods of blood famine the more remote peripheral structures of the retina had undergone some organic change. Moreover, the fields of the same patients when taken some weeks after preserved their original characteristics as regards shape and extent. Their outline was remarkable for extreme regularity of contour; no marked disparity in the affection of either halves. The degree of retraction was, however, very variable in individual instances.

Pulsation was fairly common in the retinal veins. In one case homonymous hemianopsia of a fleeting type occurred. It may be mentioned that Lodge, of Bradford, previous to my publishing these results, had reported the same constriction of visual fields in Raynaud's disease. Loss of tissue, notably in the ears and tips of fingers, may occur in rheumatoid patients as a result of these syncopes and asphyxias; while loss of nails is fairly common from the same process. I have also seen cases in which the term symmetrical gangrene was applicable: bullæ forming over the affected area, attended on their rupture by a sanious discharge, leaving a blackened underlying surface.

In one advanced case of rheumatoid arthritis the tissues over both heels underwent this necrotic change, the ulceration spreading symmetrically upwards on the outer side of both legs nearly as far as the calf, and being preceded by extreme pain over these areas.

In one instance, which happened in a woman, 35 years of age, who suffered with these local syncopes and local asphyxias, she also experienced attacks of lumbar pain coming on, especially in the day time. They were attended by a feeling of "chilliness," and a constant desire to make water, which, when passed, would be, as she termed it, quite brick-red in colour, and in a few hours would become quite clear again. On examination, the urine was of a sherry-brown colour, and microscopically it showed the presence of a few blood corpuscles in the sediment, while the supernatant fluid showed the band of oxyhæmoglobin. This case is interesting because of the association deemed by some to exist between Raynaud's disease and hæmoglobinuria. Consideration of the above facts seems to me to indicate that the vasomotor phenomena met with in rheumatoid arthritis are closely allied to, if not clinically identical with, those of Raynaud's disease. Symes[14] more recently, in his work, entitled "Rheumatic Diseases," notes this association of Raynaud's disease with rheumatoid arthritis, and has also observed the constriction of the visual fields, which he illustrates in his book by a perimetric chart showing this retraction.

SENSORY PHENOMENA.

Cases of rheumatoid arthritis differ very greatly as regards the severity of the pain experienced.

As Osler[15] says, "Pain is an extremely variable symptom; some cases proceed to the most extreme deformity without it, and in others the suffering is very great, especially at night and during exacerbations."

Again, cases in which pain is a prominent symptom are not necessarily those in which muscular wasting is a marked feature. Indeed, as Spender pointed out, "pain and atrophy are not in any definite proportion to each other. No degree of pain at the onset indicates there will be atrophy later, and the worst atrophy may be unaccompanied by pain."

As pointed out in the chapter on "Premonitory Symptoms," there are several types of pain in rheumatoid arthritis.

Articular pain of a dull gnawing character may be present, often unaccompanied by any objective tenderness of the affected joint, except where extensive formation of villi takes place, when there may be localized tenderness.

Pain is not always severe in cases presenting considerable synovial effusion; on the contrary, in instances with very little swelling, patients may complain greatly of pain, especially on movement, presumably due to attrition of the diseased cartilaginous surfaces.

Such articular pains are usually worse at night, and aggravated by warmth.

In addition, pain due to muscular cramps may cause intense suffering, in part referable to the traction exerted upon the diseased articular ends, and in part to compression of the intramuscular nerve-endings.

Finally, we have pains of a neuralgic character. One peculiarity of these pains, which stream along the length of the limb, is the difficulty experienced by others, as well as myself, in associating their path in all cases with that of any recognized nerve trunk. On the other hand, a study of these pains as they occur in the premonitory and early stages of rheumatoid arthritis seems to indicate that the site of the pain varies according to the position of the articular lesion.

Into their variations in this respect we have already entered; we have likewise pointed out the tendency displayed by them, as well as the articular lesions, to conform to a segmental distribution. It must, however, be remembered that occasionally persistent and severe pain may follow the track of the nerve trunk; and, when accompanied by tenderness to percussion and other signs of neuritis, the possibility of an underlying inflammatory process must be borne in mind. When the temporo-maxillary joint, for instance, is attacked, pain radiating over the trigeminal area on that side will be met with, which is attended, moreover, by hyperæsthesia of the skin over the same area, followed by fleeting analgesia. In such cases the conjunctiva on the same side becomes reddened, dry, and feels gritty; in some instances lachrymation and intense blepharospasm may be present. In addition, œdema of the subcutaneous tissues, extending from the nasolabial fold over the malar bone, involving the lower eyelid, may be observed.

The objective sensory modifications found in rheumatoid arthritis are usually those of hyperæsthesia followed by temporary dulling of sensation over the same area, which only in rare cases is replaced by fixed anæsthesia.

Spender noted that herpetic eruptions may follow paroxysmal pains. I have also observed this sequence in both upper and lower limbs.

SUBCUTANEOUS NODULES.

That fibroid nodules occur in this disease has been noted by Duckworth[16] and others. They are situated in the subcutaneous tissues, and, as a rule, in the neighbourhood of affected joints. I have seen symmetrical fibroid nodules along the extensor tendons of both hands; and McCrae[17] also noted a case in which the enlargement seemed to be associated with the tendons.

In a series of 110 cases he found 7·4 per cent presented these nodules; remarking that this is a much higher percentage than was noted in his cases of acute rheumatism, namely 1·5 per cent.

These nodules histologically consist of round-cell tissue, probably due to an endarteritis. Pitt[18] noted extensive thickening rather than nodules in one case marked by peripheral asphyxia. I have never seen them in children the subjects of rheumatoid arthritis. As far as my experience goes they occur in young adults, contrasting in this respect with acute rheumatism, in which the cases showing fibroid nodules are usually children.

According to Dr. Garrod[19] they are more persistent than those of acute rheumatism and more tender to the touch. Sometimes nodules are to be felt in the plantar and palmar fasciæ, and in the former especially they may be productive of much discomfort.

I have seen a row of them along the outer side of the heel resembling a rosary. In this patient also there was present a nodule situated in the palmar region of the flexor tendon of the ring finger. According to Lovett and Bradford[20] the subcutaneous tissues and fasciæ of the hand may undergo swelling, followed by thickening and contraction, which may cause flexion of the ulnar fingers as described by Dupuytren.

GLANDULAR ENLARGEMENTS.

Enlargement of the lymphatic glands occurs in quite a number of cases of rheumatoid arthritis of the adult type, and not only in that variety occurring in children, known as "Still's disease." Many seem inclined to differentiate this last-named type from all other forms of the disease. Goldthwait, indeed, apparently would go further in the process of cleavage, and would include these, as well as adult cases showing similar lymphatic enlargement, in his group of infectious arthritis.

Whether the present state of our knowledge warrants this inclusion or not, it must be admitted that this glandular enlargement is one of the most striking facts in favour of the infective origin of some cases of rheumatoid arthritis. The epitrochlear gland in emaciated cases may be not only palpably but visibly enlarged, and it is not uncommon to find a chain of glands linking it up with the axillary glands. The epitrochlear gland is the only one I have seen attain a conspicuous size. Both the inguinal and axillary groups may become enlarged, and I have also met with swelling of the suboccipital glands. I am of opinion that this glandular enlargement is frequently overlooked, and its presence in acute cases is far more constant than we are sometimes led to suppose. McCrae, in a series of 33 cases, in which special attention was directed to this point, found "general glandular enlargement in 13, definite enlargement of some of the glands in 4, and no general enlargement in 16."

Enlargement of the spleen has also been noted in adult cases. McCrae has met with four instances in which this was so.

I have never been able to detect any increase in the size of this organ in adult subjects of the affection.

BLOOD.

The first observation on the state of the blood in this disease was made by Musgrave, who, in his treatise, entitled, "Arthritis ex Chlorosi," remarks that it forms a buffy clot: a statement in which he was confirmed by Charcot as regards the acute forms. Sir Alfred Garrod also in the acute forms noted this buffy appearance in the clot due to an excess of fibrin.

The serum he found to be of normal specific gravity, and he was unable to detect uric acid or urate of soda in it.

Bocker[21] noted a great increase of calcium phosphate in the blood, this being correlated with a notable decrease in the normal proportions of this salt in the urine of the same case. Subsequently, various writers have remarked upon the pallid or anæmic appearance presented by many subjects of the malady; but for the most part their observations were not checked by the results of microscopical examination of the blood.

Forsbrooke[22] was one of the first who sought to ascertain whether this anæmic appearance was associated with any alteration in the number, or hæmoglobin value of, the red corpuscles. From his researches he claimed that a decrease occurs in the number of red blood corpuscles, ranging from 77 per cent to 52 per cent of their normal number, while their percentage of hæmoglobin was reduced from 55 per cent to 25 per cent of the normal. Subsequently, Bannatyne[23] claimed that anæmia in some degree was almost universal in this condition, finding a slight degree of it present in 95 per cent of his cases. Unlike Forsbrooke, who claimed that the anæmia preceded the onset of the disease, he regarded it as of the secondary or symptomatic type, especially marked in the very acute or advanced cases. In his series the red blood corpuscles count ranged from 4,300,000 to 3,000,000, and the hæmoglobin from 80 to 40 per cent of the normal. A slight leucocytosis was always present, this, apparently, quite independently of the anæmia or the severity of the disease.

The findings of these observers are not in accord with the results more recently obtained by Erving.[24]

This observer took a series of 20 cases, in all of which the disease was in an active stage, the determinations being made in some instances at the onset of the disease, in others during an exacerbation which occurred months, or years, after the disease had commenced.

He summarizes his results as follows :—

Red blood corpuscles—highest, 5,980,000 ; lowest, 4,148,000 ; average, 5,112,000.

Average of 17 female cases, 5,082,000.

Hæmoglobin—highest, 100 per cent ; lowest, 80 per cent ; average, 94 per cent.

Leucocytes—highest, 15,800 ; lowest, 5,400 ; average, 8,885.

Leucocytes—differential :—

Polymorphonuclear neutrophiles—highest, 80·5 per cent ; lowest, 52 per cent ; average, 64·2 per cent. Small mononuclears—highest, 40 per cent ; lowest, 17·6 per cent ; average, 27·3 per cent. Large mononuclears—highest, 12·6 per cent ; lowest, 0·8 per cent ; average, 6 per cent.

Transitionals—highest, 4·4 per cent ; lowest, absent in 4 cases ; average, 1·5 per cent.

Eosinophiles—highest, 2·8 per cent ; lowest, absent 7 cases ; average, 0·7 per cent.

Basophiles—highest, 0·6 per cent ; lowest, absent 17 cases ; average, 0·05 per cent.

McCrae, again, in a series of 33 cases, found the average percentage of hæmoglobin to be 70·6 ; while the red cells in 29 cases numbered 4,468,000. The average leucocyte count in 33 cases was 7,600. In only 9 was it over 10,000, and in 2 of these it was probably due to other conditions ; in 1 of them, during an acute pleurisy, the leucocyte count rose to 18,000 ; and in the other, the subject of a general pneumococcal infection, it reached 15,000. These two counts, however, were not included in the average. It is noteworthy, and somewhat unexpected, to find that in only 2 of the remaining 7 cases were the counts taken during the progress of an acute attack.

In 6 cases the leucocyte count was 5,000, or lower. In 8 cases full differential estimations were made, giving the following averages :—

Polymorphonuclear, 76 per cent ; small mononuclears, 14 per cent ; large mononuclears and transitionals, 8 per cent ; eosinophiles, 1·5 per cent ; mast-cells, 0·5 per cent.

Both these observers, although working independently, seem to have been struck by the discrepancy between the pallid appearances of their patients and the actual results obtained by blood examination, the findings in both their series presenting no marked variations from the normal. This tends to support Goldthwait's statement that the blood in rheumatoid—or atrophic arthritis, as he terms it—usually presents nothing abnormal. There may be a slight anæmia, he says, but this is not marked.

Teissier[25] on this point states that the number of red blood corpuscles rapidly undergoes diminution, down even to 2,000,000; while the hæmoglobin value remains normal, with some slight increase in the eosinophiles. He also notes that he has never been able to obtain cultures from the blood.

In one of my own cases, a young woman, the red blood corpuscles dropped to 2,900,000, the hæmoglobin to 30 per cent; poikilocytosis was marked. This case, however, carries very little weight, as the patient had suffered for years from gastric ulcer, and on two occasions had hæmatemesis.

Waterhouse and Bullmore,[26] as a result of examination of 42 cases in an active phase of the disease, came to the conclusions that anæmia was almost always present in rheumatoid arthritis; the number of red blood corpuscles sank to between 75 per cent and 95 per cent, the hæmoglobin value sinking a little lower to between 70 per cent and 90 per cent. They noted that a leucocytosis was not present in the great majority of cases, neither were the normal proportions of the different varieties in any way altered.

Josh. R. Billings, Jun., recorded a case of rheumatoid arthritis in which the blood changes characteristic of pernicious anæmia were found.

In reviewing the results obtained by these various observers it is obvious that no constant changes are to be found in the blood in these cases, which is somewhat surprising when one considers the severity of the malady and the wasted, sallow, and apparently anæmic appearance presented by the sufferers from it.

CONSTITUTIONAL SYMPTOMS.

Pyrexia.—Both in the acute and subacute varieties the body temperature is raised. In the former it is of comparatively high grade, continuous, and presents a very irregular curve.

Temperatures of 103° are not uncommon, and Bannatyne has recorded one instance in which 105° F. was reached. Such a condition of pyrexia may continue for weeks, and even months. In some instances rigors have been noted. Ultimately, this continued pyrexia undergoes remission, to be succeeded by a series of exacerbations with intervening apyrexial periods of increasing length.

In the subacute forms the pyrexia which accompanies their onset is of much shorter duration, and does not remain above normal for more than a few days, when it declines, and, pending the involvement of fresh joints, may remain so.

Extension, however, of the disease is attended by another rise of temperature; thus we get an approximation to what is obtained in the later stages of more acute forms; namely, a series of periods of pyrexia followed by periods of apyrexia. The temperature in such attacks as a rule ranges between 99° and 101° F.

Finally, in those cases of gradual insidious onset the temperature may never rise throughout the disease, though the arthritic changes may be just as severe as those in which the temperature is raised.

This subject of pyrexia in rheumatoid arthritis is accorded a much more prominent place in the descriptions of some writers than others. Some, like Bannatyne, maintain that a condition of pyrexia is far more common than is usually supposed; others, like Goldthwait, apparently consider that the disease runs an apyrexial course. It is possible that this discrepancy between these two observers is due to the fact that some, like Goldthwait, do not recognize the term "acute rheumatoid arthritis," and are wont to assign the types designated so by English authorities to the category termed by them "infectious arthritis."

The abstraction of the more acute forms with glandular enlargements would necessarily much diminish the number of cases in which pyrexia was found. The same difference on this point obtains between the statements of Teissier and McCrae.

The former maintains that there is no pyrexia, while the latter, in a series of 86 cases, found that the temperature was raised in the great majority. More extended observations are necessary before this question can be placed on a satisfactory basis, and in all probability these differences in opinion will continue until the lines of delimitation between the more acute forms of rheumatoid arthritis and the infective arthritides are more clearly ascertained.

CIRCULATORY DISTURBANCES.

Increased frequency of the pulse-rate has long been recognized as a feature of this disease. Charcot noted that the pulse was evidently accelerated ; and I think, if we may judge from the following quotation from his Clinical Lectures, Graves also had noticed this undue frequency in the heart beats. Thus, he says, "Cases of rheumatic arthritis, attended with prolonged excitement of the circulation and copious sweating, are generally found to exhibit an intractable chronicity, and too often terminate in rendering the unfortunate patient a cripple for life."

As may be gathered from his writings on this type of disease, which he termed "sweating arthritis," he attached grave prognostic significance to constant acceleration of the pulse, which he says "is always a bad sign." Duckworth also noted that palpitation was not an infrequent feature in this disease. It is, however, to Spender that we are indebted for the primary recognition of this now well-ascertained characteristic of the disease.

This authority claimed that in 80 per cent of his cases the pulse-rate was found to be more than 90. In many instances it ran up to 110, 120, and in one case reached 140 beats per minute. He noted that it occurred with a normal temperature, and could, therefore, not be regarded as a simple concomitant of pyrexia. Furthermore, it was not associated with any disease of the heart or œdema of the extremities. He was inclined to attribute it to a diminished inhibition on the part of the pneumogastric nerve. Of the substantial accuracy of this clinical fact there can, I think, be little doubt ; and in isolated instances I can quite confirm him that it can run up to 140, and even higher.

It is, perhaps, not so well recognized that Spender attached great importance to examination of the condition of the thyroid gland in rheumatoid patients. He himself noted an instance of this tachycardia with coincident enlargement of this structure in a case of rheumatoid arthritis ; and, furthermore, suggested that there were points of affinity between exophthalmic goitre and this malady. In a later section I shall refer more in detail to this suggested association between the two conditions and its possible relationship to the quick pulse of rheumatoid arthritis. In conclusion, I would lay stress upon this quickened pulse as a substantial aid to diagnosis, and, furthermore, on its persistence as of grave omen.

SWEATING.

Profuse general sweats are a very constant feature in the acute cases ; at later periods, however, they tend to become localized to certain areas. They may be confined to the lower or the upper extremities, or even to portions of them. Thus localized sweating of the dorsum of the foot and the neighbouring portion of the leg is very noticeable when the clothes are stripped off a bedridden rheumatoid patient. Over the back of the forearms such may be observed. Such localized sweats may follow the decline of neuralgic paroxysms in any particular area, or in sequence to vasomotor disturbances. Similar limited perspiration may follow trigeminal neuralgia when the disease is in an acute stage in the temporo-maxillary joint. The plantar and palmar surfaces are the areas which most frequently display this phenomenon, the palm and sole running with moisture, though they feel cold and deathlike to the touch, while they are semi-asphyxial or unnaturally white in colour.

CUTANEOUS CHANGES.

In the early stages of rheumatoid arthritis the main changes in the skin are those due to abnormal pigmentation and those resulting from excessive secretion of the sweat glands. This pigmentation was inimitably described by Kent Spender. It varies widely as to degree in different cases : in some cases it is very marked and widespread, arresting the attention immediately by its conspicuousness. The following vivid description is taken from the writings of Dr. Spender :—

" Concentrated in patches more or less large, the pigment assumes many hues, and affects many parts of the body. Across the forehead it spreads as a light bronze smear, or like a dash of chloasma ; over the temporal fossa on each side the tint may be deeper. Under the lower eyelids the streak of colour may be very dark, and may shine with metallic polish. The dominating tints on the face are lemon, orange, and citron ; the lustre varies with the angle of reflected light. In people of dark complexion the colour of the face is sometimes so swarthy as to make the patient resemble a mulatto. The white of eye stands out in brilliant contrast; and in some cases the neck looks as if soaked in walnut dye; beginning at the line at which it is ordinarily covered the skin has the appearance of being unwashed."

While cases showing the widespread pigmentation of the region of the face and neck do occur, it is often much less marked, or entirely absent. The same erratic tendency is displayed by the so-called Spender's spots. These, as the above authority pointed out, take the form of spots scattered apparently at random over the forearms, legs, hands, and feet, and are to be occasionally seen in the neck. They are of a brownish or blackish-yellow colour, and are not raised above the surrounding skin.

Although, as a rule, it is not possible to associate their area of distribution with any particular nerve, I have known them to appear in the intercostal spaces in sequence to severe neuralgic pains in that situation; and, in like manner, they may follow neuralgic pains of the forearm and hand. Spender also points out that in cases of obscure neuralgia of the lower limb the appearance of "odd yellow blotches" along its continuity may be taken as an indication of oncoming rheumatoid changes in the joints of the affected extremities. The similarity of the pigmentation, both as regards its character and distribution, to that obtaining in Graves's disease, was noted by Spender; and I may mention that some examples under my own care showing marked pigmentary display were the subjects likewise of exophthalmic goitre.

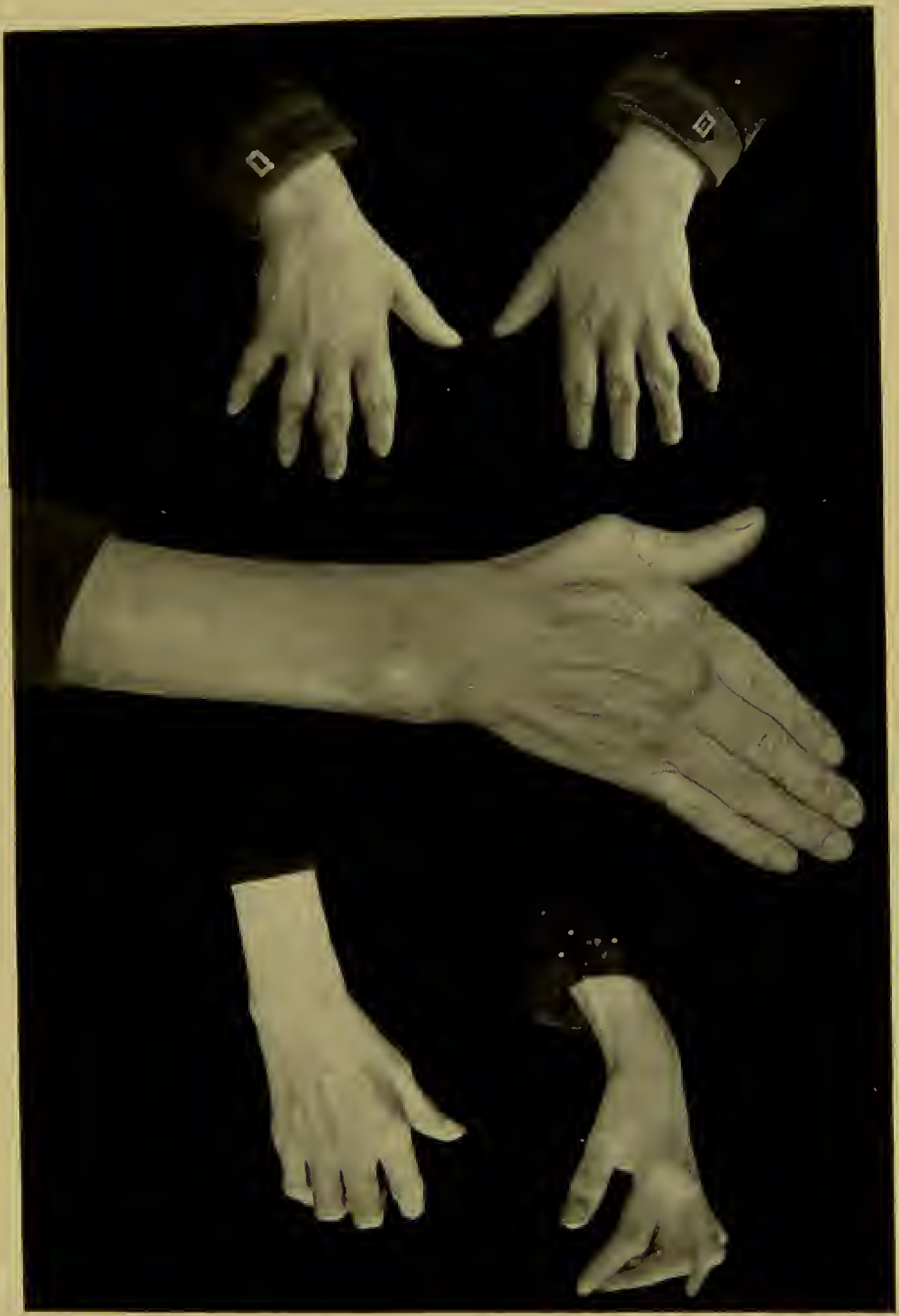
TERMINAL STAGE OR SECONDARY CHANGES.

Hitherto we have been concerned chiefly with the disease in its more active phase, a period during which many of the phenomena are of a fleeting or ephemeral type. In the more favourable cases the periarticular swellings may rise and wane almost to abolition, the sensory perversions are transient and swiftly pass away, and the vasomotor phenomena may leave no trace, while the muscular irritability may decline, and the muscular atrophy stop short of contracture. Unfortunately, in all too many instances, the clinical picture at this stage takes on more sombre hues, and this hope-enlivened phase gives place to the settled gloom of structural alteration. What we may term the "pathological habit" crystallizes into a fixed attitude, and we are confronted with a series of morbid changes, the legacies, so to speak, of the foregoing pathological processes.

The hitherto swollen and spindle-shaped joint begins at this time to undergo a process of gradual shrinkage, becoming smaller and smaller (*see Plate V, lower figure*), until eventually in the more protracted and severe forms it becomes reduced even below its normal size, forming, as Goldthwait terms it, the small "end-joint" of atrophic or rheumatoid arthritis. All the articular structures participate in this atrophic change, and, according to Goldthwait, even at the very beginning of the disease some degree of absorption of the cartilage may be demonstrated by skiagraphy. The shafts of the bones participate in the atrophic process, and when, as not infrequently happens, the ends of the bones telescope into each other, a distinct decrease in length of the affected fingers may take place. The vasomotor phenomena, so frequent in the prodromal and active phases of the disease, tend gradually to disappear; and in the meantime, possibly as a result of these circulatory disturbances, the skin, especially over the extremities, begins to show signs of impaired nutrition. These secondary changes are best observed in the fingers and hands (*Plate VII, right-hand figure*).

The skin over the palm, previously thickened and soddened with perspiration, becomes unnaturally dry and harsh, desquamates freely, and loses its suppleness. Finally, in the late stages, it becomes obviously thinned, and of a pinkish-white hue. The skin on the fingers undergoes similar changes, becoming smooth, thin, and bluish-white in colour, while the wrinkles over the joints become diminished in number and size. Often, at this stage, the joints appear tucked in, and any attempt at flexion is checked by the shrunken integument. When, as a result of muscular contracture, the fingers are in a state of permanent flexion, the skin over the articulations may become so thin as to appear almost diaphanous. These atrophic changes are not confined to the periphery of the extremities, but such may be met with on the skin of the limbs—in my experience, most

PLATE VI.

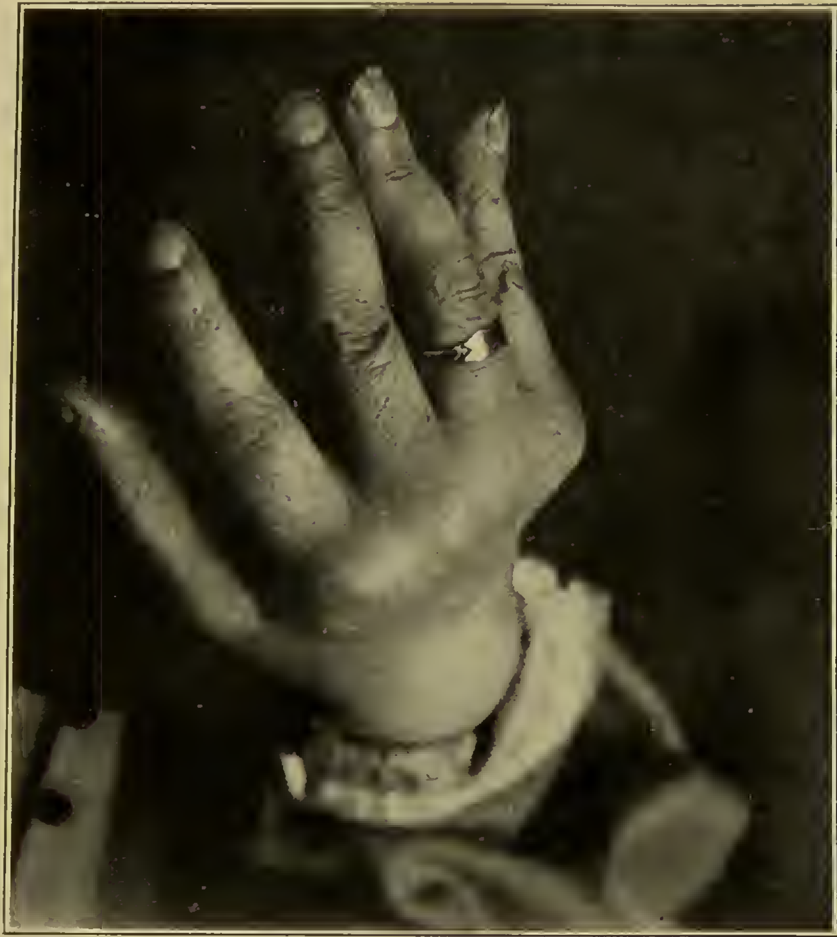


Upper Figure:—Very irregular swelling of mid-phalangeal joints due to hernial protrusions of synovial membrane.

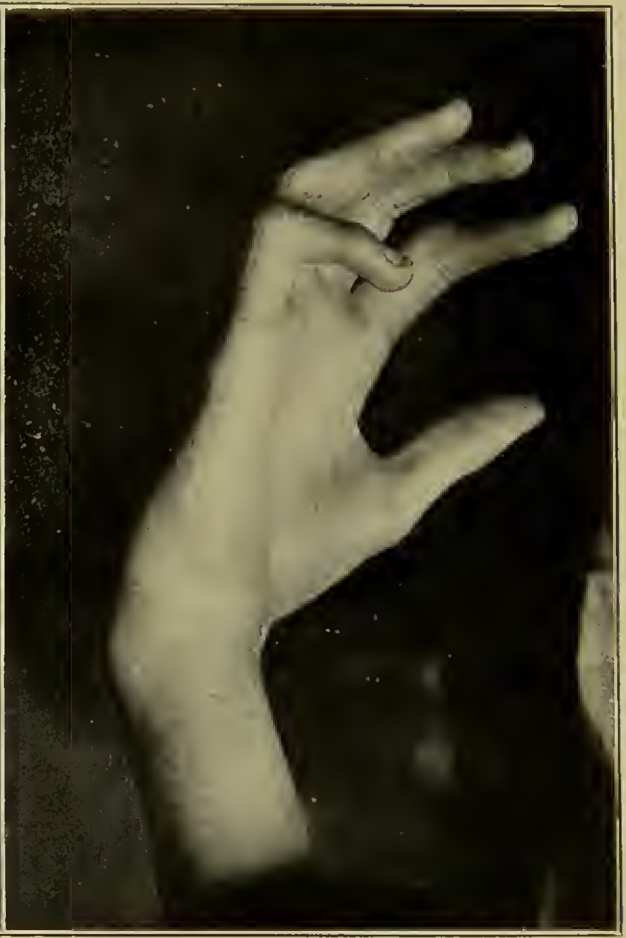
Centre Figure:—Ulnar declination of fingers.

Lower Figure:—Fixation of wrists in straight line, with permanent flexion of fingers.

PLATE VII.

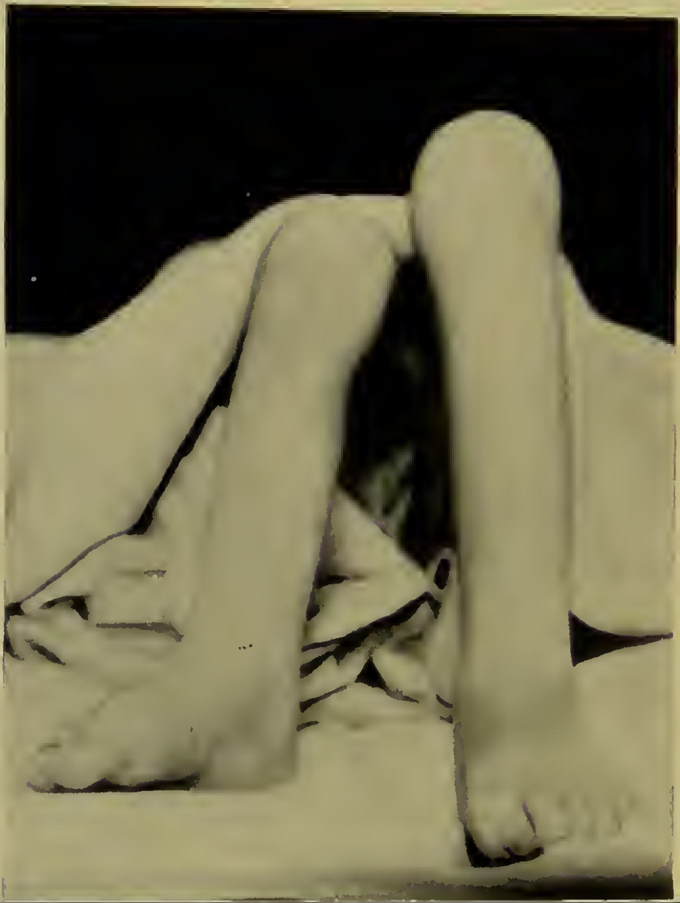


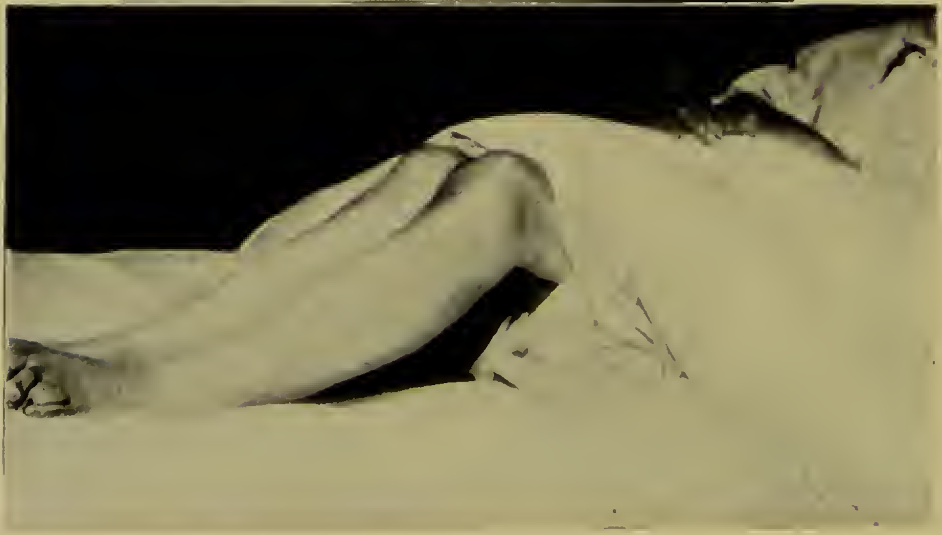
Left Figure. Fixation of Wrist in a position of extension, with dislocation backwards of mid-phalanx of middle finger



Right Figure.—Hand of subject of rheumatoid arthritis with severe Raynaud's disease, followed by atrophy of skin over palmar surface of hand and fingers.

PLATE VIII.





Upper Figure. Showing œdema of the lower extremities.
Lower Figure.—Note glossy atrophic condition of skin on outer side of leg.

commonly on the dorsum of the forearm and outer side of the leg. Over these sites in the earlier stages I have observed localized sweats, also an overgrowth of hair of recent date.

In some instances, owing to the wasting of the subcutaneous tissues, the skin feels tied down to the bone, and as the change, in some cases, may involve the dorsum of the foot as well as the whole of the leg, the impression given on grasping the limb is similar to that experienced when the hand is laid on the limb of a statue.

It is in cases, however, where the condition is less general, and confined to the outer side of the leg, that we are best able to appreciate the departures from the normal by comparing them with contiguous areas. The area of disorganized skin may more or less accurately follow the territory supplied by the fifth lumbar segment in the leg and the eighth cervical in the forearm. The skin over this region in the later stages may lose the overgrowth of hair before alluded to, and its smooth, ivory-white appearance suggests a condition of scleroderma (*Plate VIII*). While neighbouring areas sweat, this region remains perfectly dry, with a lowered surface temperature, and may show marked sensory alterations of a permanent character. I have seen fixed anæsthesia in such a case. Teissier also has noted these secretory and sensory alterations over areas of skin thus affected.

As opposed to this secondary atrophic change, which, according to Charcot, Vidal, and Teissier, is more common in the upper limbs, we have now to consider the reverse condition, enlargement of the limbs, which, on the contrary, is more common in the lower extremities. As Charcot long since pointed out, the lower extremities in some cases become the seat of an œdematous infiltration simulating, in his opinion, elephantiasis. The œdema is, as a rule, confined to the leg and foot; it is solid in character, does not pit on pressure, and occurs independently of renal and cardiac troubles.

The overlying skin is harsh and dry; but I have seen the integument in such an œdematous leg take on the atrophic characters above described. According to Teissier, the same disorders of sensibility, with the same lowered surface-temperature, are to be met with as in the preceding atrophic forms.

Diamantberger and Moncorvo[27] have also noticed this secondary œdematous condition; the former claims that, objectively at any rate, it is, though localized, indistinguishable from the diffuse swelling of myxœdema.

It now only remains for us to allude to a peculiarly malign ulceration of perforating character which sometimes makes its appearance in this affection. According to Macalister,[28] of Liverpool, it constitutes one of the most grave complications of rheumatoid arthritis, inasmuch as the general septic infection, which almost inevitably follows, puts an end to the patient's life. He records six instances in which the death of the patients appeared to be directly related to this formidable local necrosis. In one example the inner side of the knee was the site, and perforation of the joint cavity followed. In three cases the ulcer appeared on the dorsum of the foot; and in one of these instances the limb became gangrenous; and, in spite of amputation, death ensued. In two instances they resembled the bed-sores in myelitis, in being situated over the sacrum; the ulcers were deep, with gangrenous surroundings, and death ensued.

MUSCULAR CONTRACTURES.

The forced attitudes assumed by the limbs as the result of muscular spasm are in the earlier stages more or less temporary.

In the more advanced periods atrophy gives way to contracture, and permanent distortion results. The deformities resulting from this are, in my experience, more pronounced in young people; and the disease here contrasts markedly with osteo-arthritis, in that while in the latter there is deformity of the joint due to irregular bony outgrowths in the line of the articulation, in the former there is no deformity of the joint, but a distortion in the position of the limb, the different parts of which undergo an altered relationship to each other. In other words, distortion of the limbs is the distinctive characteristic of rheumatoid arthritis, and deformity of the joints of osteo-arthritis.

These malpositions have been closely studied by Charcot, who divided those of the upper limb into two chief varieties, of which the *first type* is the most common; namely, that in which flexion predominates.

It is characterized by :—

1. Flexion of the ungual phalanx on the second phalanx to an obtuse, right, or even acute angle.

2. Extension of the second phalanx on the first phalanx (*see Plate VII, left figure*).

3. Flexion of the first phalanx on the metacarpal bone.

4. Flexion to a less obtuse angle of the metacarpus and carpus on the bones of the forearm.

5. In a great many cases ulnar declination of all the fingers, and deflexion in the opposite direction of the second phalanges on the first.

There are two variations from this type. In one, the first and second phalanges are in the same axis and form a single column. In the second sub-variety there is no flexion of the ungual on the second phalanx, and the backs of the fingers are hollowed out, presenting a dorsal concavity.

The *second type* is characterized by :—

1. Extension of the ungual phalanx on the second phalanx.

2. Flexion of the second phalanx on the first (*see Plate VI, lower figure*).

3. Extension of the first phalanx on the metacarpal bone.

4. More or less flexion of the carpus on the bones of the forearm.

5. Occasional deviation of all the fingers towards the ulnar border of the hand.

In this type, as in the former, there are two sub-varieties.

In the first there is flexion of all the segments of the hand on one another, so as to appear rolled up.

In the second the same characters are found, and there is, besides, extension of the second phalanx on the first. The thumb participates in the malpositions : its proximal phalanx is usually flexed, occasionally extended. The elbow joint is fixed in a position of flexion, the forearm being pronated, while the carpus and metacarpus are more or less flexed upon it. In cases where the shoulder is involved, the upper arm may be closely adducted to the chest. In the lower limbs, while the mobility of the hips is, as a rule, spared, the knee undergoes great distortion. According to Charcot :—

1. The lower end of the femur projects in front of the head of the tibia.

2. The internal condyle becomes more prominent.
3. The patella, thrown outwards, rests on the outer condyle.
4. The head of the fibula projects externally.

The foot is, as a rule, abducted, and rests on its inner edge, resembling in many respects talipes valgus. It may, on the contrary, be adducted, and give rise to an equino-varus.

The big toe is displaced outwards, covering the other toes of the foot. This condition of hallux valgus may be associated with hammer-toes.

These deformities, so laboriously studied by Charcot, are, however, I think, too rigidly defined, for, especially in the different fingers, as Teissier points out, the separate types may be represented, and thus overlapping occurs.

Opinions have differed greatly as to the mode of production of these various malpositions. Among the older physicians Beau[29] attributed them to the voluntary attitudes assumed by the patient to lessen the intensity of the pain; and Trastour[30] held a similar opinion. Charcot and Crocq, however, came to the conclusion that the main agent concerned was muscular spasm, basing their argument on the fact that the attitudes assumed are forced, and not such as will be produced by voluntary action. In addition, these same malpositions are assumed in other diseases in which the joints are not involved. They are met with in paralysis agitans, atrophy of the interossei, and other diseases in which the joints are in no way affected.

It is recognized nowadays that they are the consequence of a condition of muscular imbalance, the atrophy, with consequent weakness of certain muscles, leading to overaction of their opponents; thus, in the knee-joint, the wasted quadriceps extensor is more than overcome by its less wasted antagonists, the hamstrings. Certain factors, such as the weight of the limbs, and a more or less lax condition of the ligaments, contribute to supplement the above fundamental cause of the distortions. The contractures in their most aggravated form give rise to subluxation or dislocation of the bones entering into the articulations. This may be seen very plainly in the wrist, where the distal ends of the radius and ulna project backward, while the carpus sinks forward.

In the knee, as the result of the predominance of the flexor muscles, subluxation of the head of the tibia backwards takes place, leading to marked prominence of the condylar ends of the femur. The patella, as mentioned before, tends to be dislocated outwards. In the fingers undue prominence of the heads of the metacarpals will result from subluxation forward of the bases of the proximal phalanges.

In the terminal stages of the disease the cartilage may undergo complete absorption, and the ends of the bones sharing in the destructive process may telescope one into the other, as shown by X rays. As a result, the irremediable nature of the deformity brings about complete immobility, and direct bony ankylosis may result.

VISCERAL MANIFESTATIONS.

Cardiac Complications.—Considering the acute course run by the disease in some instances, and the severity of the articular lesions, coupled with the many indications it presents of an infective or toxic origin, it is remarkable how seldom there are signs of coincident endo- or pericardial involvement. I have never in my personal experience observed endocarditis arise *de novo* in the course of even the acuter forms ; but I have seen pleurisy without effusion occur.

In a series of 126 cases taken in succession out of one of my case-books, I found 15 presented evidences of cardiac disease, implicating in 2 both aortic and mitral orifices, while of the remainder, 1 was a case of double aortic disease, and 12 were examples of mitral disease, 2 of which were uncomplicated stenosis.

These results approximate to those of McCrae, whose investigations into the cardiac condition of 74 cases afforded the following data : In 55 there were no signs of disease ; in 10 there was a soft systolic bruit, apparently functional ; in 7 there were present organic lesions ; 3 of aortic insufficiency ; 2 of combined mitral stenosis and insufficiency ; and 1 each of mitral insufficiency and mitral stenosis. In some of my own series there was a history of previous repeated attacks of a polyarthritis. In one there was a history of chorea in addition.

It is difficult in such cases to exclude the possibility of the endocardial lesions being due to a previous attack of acute rheumatism. I say this, though fully aware that some would hold that the previous attacks were manifestations of acute rheumatoid arthritis. I cannot help feeling that we go too far in assuming that these previous joint affections must inevitably have been rheumatoidal in character: as if there were something antagonistic in the soil which precluded the possibility of the patient suffering at an earlier period of his life from acute rheumatism. The case I have quoted above of polyarthritis preceded at some distance by chorea would seem to indicate the possibility that rheumatoid disease can, at any rate, supervene in a patient who has previously suffered from rheumatic manifestations. On the other hand, as Dr. Garrod reminds us, endocardial lesions may be produced by a variety of organisms; and there being much in the symptomatology of rheumatoid arthritis suggestive of a toxic or infective origin, it is possible yet, though not to my mind clearly established, that endocarditis may arise in the course of rheumatoid arthritis.

Gastro-intestinal Affections.—According to an increasing number of observers the subjects of rheumatoid arthritis very frequently suffer from disturbances of the digestive functions.

In many instances the alimentary derangements precede the appearance of joint swellings, while in others they apparently arise contemporaneously.

The gastric derangements may be slight or aggravated. Frequently these patients have a foul tongue and breath, and complain of a disagreeable taste in the mouth, lack of appetite, discomfort and flatulent distention after meals.

In others, the gastric symptoms are more severe, and are attended by pain after food, persistent vomiting, and rapid failure in nutrition.

I have in a few observed the so-called classical symptoms of gastric ulcer with hæmatemesis.

Grave departures from normal may be present without any subjective symptoms of dyspepsia, as demonstrated by Dr. Helen Baldwin, who, as before referred to, found an absence of free HCl after a test meal in three cases.

Spender noted the occurrence of "gastric crises," for which there was no assignable cause, dietetic or otherwise.

Excessive sluggishness of the liver, with an unhealthy condition of the intestinal secretions, is often present, the stools being either dark and extremely offensive, or else of an unnaturally pale colour.

According to Tubby, the fæces usually have an alkaline, instead of a normal acid reaction, with an exceedingly foul odour.

This observer, with others, has noted the presence of excessive amounts of mucus in the fæces, and the occasional passage of typical casts of the bowel.

Alkalinity of stools is, according to Herter, usually met with in the indolic type of chronic excessive intestinal putrefaction; and Tubby notes that, though variable, yet indican in abnormal amounts has been found in the fæces.

Constipation, as might be expected from the enforced inactivity, is not uncommon, and, according to Dr. Katherine S. Clark, may alternate with persistent and troublesome diarrhœa.

In a case recently under my own observation the patient was the subject of chronic diarrhœa, which had antedated the appearance of the joint affection; remissions and exacerbations were correlated with diminished, or increased, severity of the intestinal affection.

The disease has been known to arise in sequence to ulceration of the large intestine.

Renal and Urinary Changes.—An excess of urea and uric acid was noted by Angel Money, also transitory glycosuria; but most observers have been unable to substantiate his statement as to the excess of urea and uric acid.

Glycosuria has, however, been noted by others. Diminution in the amount of phosphates excreted was noted by Drachmans, and likewise by Bocker.

According to Teissier, the amount of urea excreted is diminished; at the onset of the malady the urotoxic co-efficient is normal, but in the more advanced stages it sinks below normal, the co-efficient ranging from 0·25 to 0·2.

In his experience albuminuria often supervenes at this stage of the malady.

Variable amounts of indican are to be found in the urine. According to Herter, excessive indicanuria was ascertained by him to be constantly present in cases of early rheumatoid arthritis.

The frequent presence of an excess of aromatic sulphates has been emphasized by several observers, notably Armstrong, of Buxton.

McCrae, as a result of an analysis of the urine of 83 cases found that in 64 secretion was normal; in 10, albumin and casts were present; while in 9 a very slight trace of albumin alone was detected. Sugar was found in 1 instance. The total secretion of uric acid was determined in a number of cases; but no departures from normal were met with.

Chronic nephritis with albuminuria was considered by Charcot, Cornil, Fuller, and others of the older writers, to be a not infrequent complication of the disease in its later stages. In my experience, however, it is of very rare occurrence.

Garrod notes that acute nephritis, accompanied by a highly albuminous urine, has occurred in isolated instances of the disease.

Teissier is of opinion that there is a marked tendency in sufferers from rheumatoid arthritis to succumb either to renal disease or tuberculosis.

Other visceral complications are those of bronchitis and pneumonia. In a case under my own observation the patient fell a victim to pneumonia.

Episcleritis, iritis, and conjunctivitis are occasionally met with. Episcleritis I have seen several examples of, generally involving both eyes, but in both this and iritis there is not infrequently a suspicion of a gonorrhœal taint.

ASSOCIATED DISEASES.

As far back as 1888 Kent Spender drew attention to points of affinity which he believed to exist between rheumatoid arthritis and exophthalmic goitre; and, when Medical Officer to the Bath Mineral Water Hospital, I had abundant opportunities of verifying the accuracy of his observations.

That this opinion of Spender was shared by others, is evidenced by the statement of Buzzard, in his article on "Tremor," in the "Encyclopædia Medica." Thus, he writes, "The four diseases—neurasthenia, rheumatoid arthritis, paralysis agitans, and Graves's disease—are purposely placed together, because they appear in more ways than one to be related, and belong to a group the pathology of which is still obscure." In continuation, he refers to the similarity of tremor in neurasthenia to that of Graves's disease, and points out that the hemiplegic or diplegic distribution of tremor found in paralysis agitans is duplicated in some cases of rheumatoid arthritis. It would appear, therefore, that the clinical features of rheumatoid arthritis seem in many ways to approximate themselves to those typical of various nervous diseases.

Passing now to the consideration, more particularly, of its relationship to Graves's disease, one notes that, apart from the occasional presence of tremor, the two diseases have further points in common, such as the quickened pulse-rate, somewhat similar disorders of pigmentation, localized sweatings, localized œdemas, etc.

Spender, in an article devoted to the consideration of these affinities, instances two sisters, one of whom had rheumatoid arthritis, the other Graves's disease. I also have seen these diseases in different members of the same family. He appears, however, only to have met with one example in which the two affections co-existed in the same patient, which he described as "an extremely crippling arthritis associated with exophthalmos and paroxysmal hurry of the heart."

The same author narrates how enlargement of the thyroid cropped up every now and then amongst his rheumatoid patients; and in the same erratic manner exophthalmos, tremor, and pigmentation, one or other of them, was present occasionally. Similarly, in the course of my hospital work, I was struck by the "staring expression" of some of the patients, and it is at any rate worth mentioning that Still, in the disease associated with his name, remarks, "a curious occasional symptom is slight prominence of the eyeballs; it is not associated with enlargement of the thyroid gland." With further observation, I came upon cases where one, two, or three of the so-called cardinal symptoms of Graves's disease were present in individual rheumatoid patients, and ultimately I came across instances where all four symptoms—exophthalmos, thyroid enlargement, tremor, and quickened pulse-rate—existed in this association.

Some years ago I reported in the *British Medical Journal* fourteen examples of the complete type of Graves's disease, and six of the larval or incomplete type, existing side by side with rheumatoid arthritis. I came to the conclusion that the relationship between these two diseases was not merely one of points of affinity, but that they might actually overlap in one and the same patient. In the fourteen examples of the complete variety, the patients were all of the female sex; their average age was 29·9, the oldest being 52 and the youngest 18.

The previous history was of a most varied character.

In one, the illness dated from her fourteenth confinement; in four, the cause assigned was influenza; and of these one, in addition, had suffered from two attacks of "rheumatism," and another from anæmia.

Of the rest—two gave a history of tonsillitis and rheumatism; one, gastric ulcer and hæmatemesis; one, scarlet fever; one, sore throat, anæmia, and ozæna; one, rheumatism; one, anæmia, indigestion, and sore throat; one, sick headache.

In two cases the symptoms of exophthalmic goitre had preceded by some years the development of rheumatoid arthritis.

In the majority, the two diseases apparently arose contemporaneously; but in one or two the symptoms of exophthalmic goitre were engrafted upon a previously-existing rheumatoid arthritis.

At the time they came under my observation the joint troubles had existed for varying periods; the minimum being three months, and the maximum two years, in duration.

In ten out of fourteen cases there were present, in addition, varying degrees of local syncope and local asphyxia of the fingers. As regards the ocular symptoms, in addition to the proptosis, the signs of Stellwag and von Graefe were very marked in some cases, in others less so. Weakness of convergence, namely Mœbius's sign, was in some easily ascertained. Slight degrees of ptosis and paresis of the external recti were noted. The thyroid swelling was usually moderate in size, and certainly varied from time to time in some of the cases. The tremor in these complete forms was, as a rule, general, but, as Buzzard states, it may be hemiplegic or diplegic in distribution. It is often more marked in the limb which is the seat of joint swellings. The highest pulse-rate that I have noted was 140; and it showed the same tendency in all cases to be increased by slight causes, as in uncomplicated cases of Graves's disease. It will be remembered that Spender, speaking of the rapid pulse of rheumatoid arthritis, claimed :—

1. That it occurs in some cases ; not in all.
2. It occurs with a normal temperature.

Both are important distinctions ; the former, because it indicates that a rapid pulse-rate is not an essential symptom of rheumatoid disease ; the latter, because it disposes of its only being found in acute forms, where it might be thought to be simply a concomitant of pyrexia. We may take it, therefore, that the quickened pulse-rate, though frequent, is not a constant feature, but tends to manifest itself in an erratic manner in cases of rheumatoid arthritis. This suggests the possibility that, in some instances, it may be a larval manifestation of Graves's disease.

Lately I saw an interesting case of the polyarticular type of rheumatoid arthritis in a young girl, who also had an enlarged thyroid, with a pulse-rate of over 120. Both her arms and hands were the seat of patches of leucoderma symmetrically arranged. She had no exophthalmos ; but following an attack of severe pain radiating along the inner side of left forearm and hand, there appeared an extensive crop of herpes, extending chiefly over the eighth cervical and first dorsal areas (Head), and coincidently there were developed signs pointing to some interference with the cervical sympathetic on the same side, namely, enophthalmos, pseudoptosis, with a contracted pupil. This symptom group may, as we know, be produced by any affection of the eighth cervical and first dorsal nerve-roots, if such lesions affect the roots at a point before they give off their rami communicantes.

I mention the case because, as far as I am aware, no similar one has been recorded in the literature.

Much interest, and possibly some profound significance, attaches itself to the fact observed by Spender and other authorities, that many of the so-called secondary features of Graves's disease occur in the subjects of rheumatoid arthritis, and this in cases where none of the cardinal symptoms of the former affection are present. Among these may be mentioned localized sweatings and fleeting œdemas of angioneurotic type; and, in addition, certain skin affections, such as leucoderma and scleroderma. The former I have several times observed on the hands, the affected areas showing a striking symmetry in arrangement; the latter is more commonly to be met with in the lower extremities.

Muscular cramps simulating closely those of tetany have been observed in both diseases, likewise transitory glycosuria. Gastro-intestinal derangements and uterine disorders are common in both.

Further instances of the overlapping of the symptoms of the two conditions might be cited, but the purpose of this section is fulfilled if it demonstrates the need of further observation on this interesting affinity of the two diseases for each other.

Macalister has also drawn attention to the obscure kinship that appears to prevail between the two affections.

Solis Cohen has reported an instance of their co-existence in the same individual.

E. I. Spriggs, at a recent meeting of the Royal Society of Medicine, exhibited a man showing the clinical features of rheumatoid arthritis and exophthalmic goitre. Through Dr. Spriggs' kindness I am enabled to show the accompanying photograph taken by Mr. Damon, also the skiagraph of the patient's hands executed by Dr. Simmons, radiographer to St. George's Hospital (*Plates IX and X*).

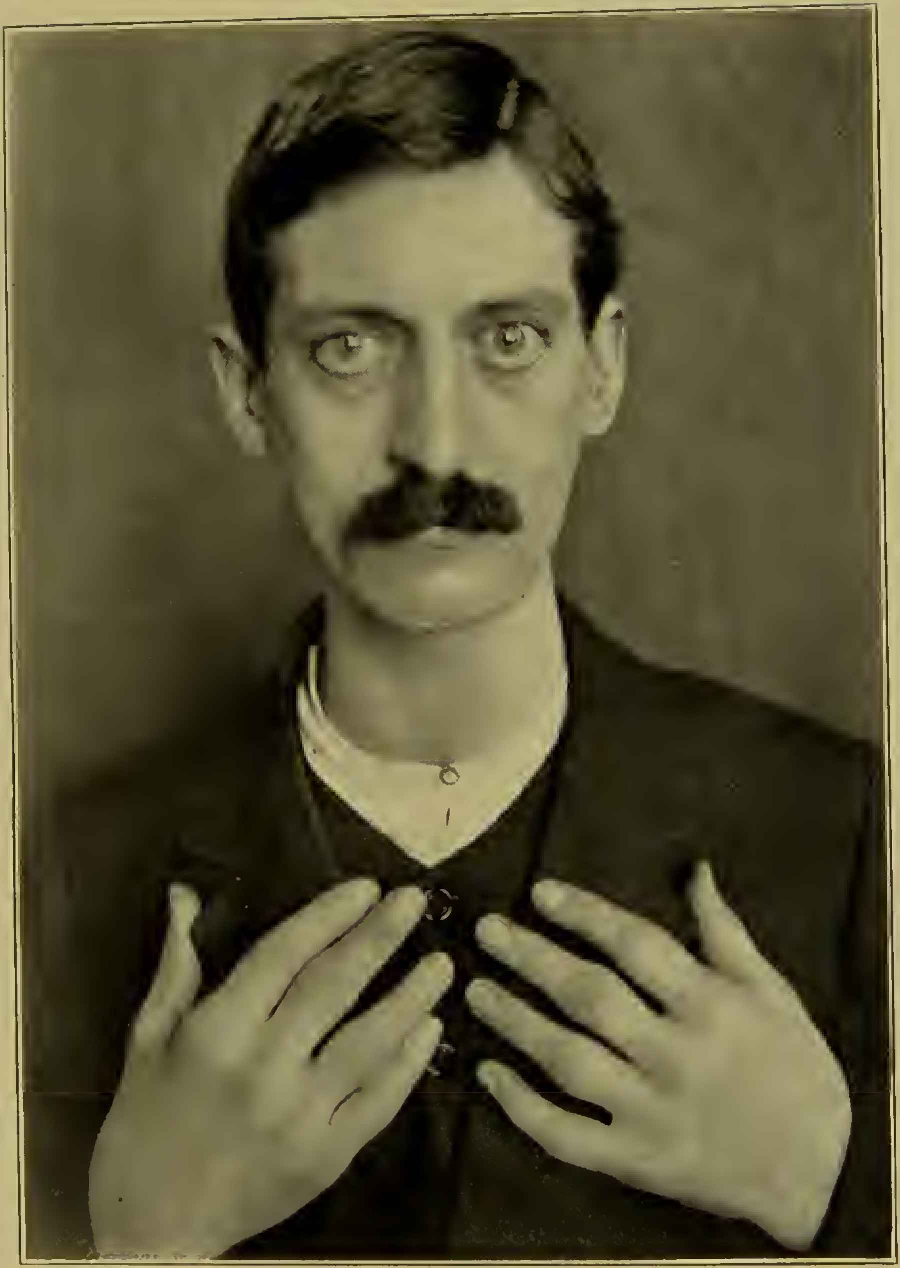
Symes, in his work on the "Rheumatic Diseases," comments on their occasional association, while Diamantberger and Weil have contributed similar views.

Unfortunately, this affinity on the part of rheumatoid arthritis for Graves's disease, though highly interesting, throws little or no light upon the mode of genesis of the former affection, the pathogeny of the latter being equally obscure.

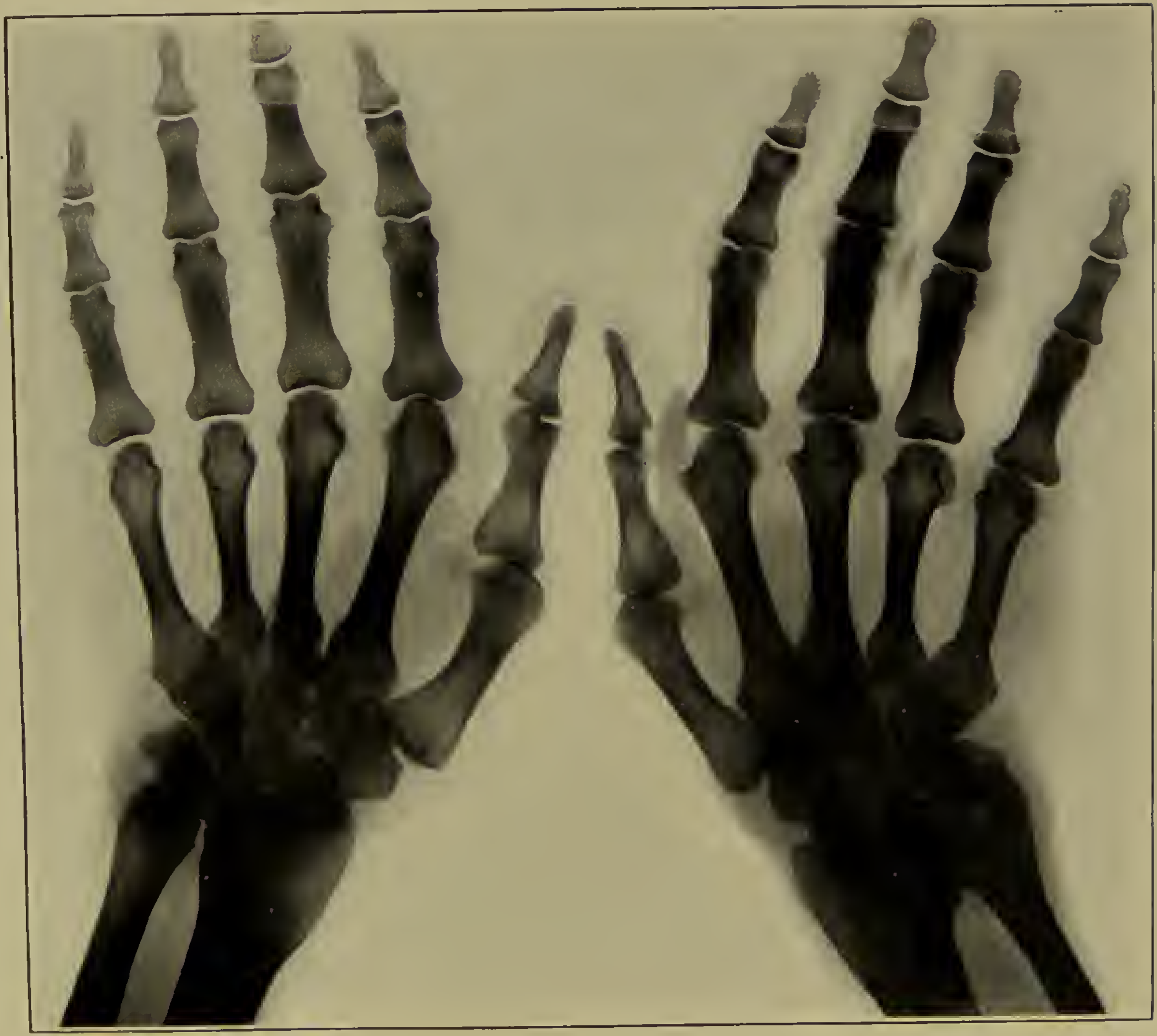
In view of the close relationship of Graves's disease to myxœdema, it is worthy of note that Macalister has recorded an instance in which the condition of myxœdema was superimposed upon that of rheumatoid arthritis. The case is such a remarkable one that I append the following brief account of its clinical history.

The patient was a woman, aged 62, who three years

PLATE IX.



Dr. Spriggs' Case of rheumatoid arthritis and exophthalmic goitre.



SKIAGRAPH OF DR. SPRIGGS' CASE, showing some enlargement of the distal ends of the proximal phalanges, with some irregularity of outline, especially of second and third fingers. There is also some irregularity of outline, and slight loss of [illegible] of the head of the second metacarpal.

PLATE XI.

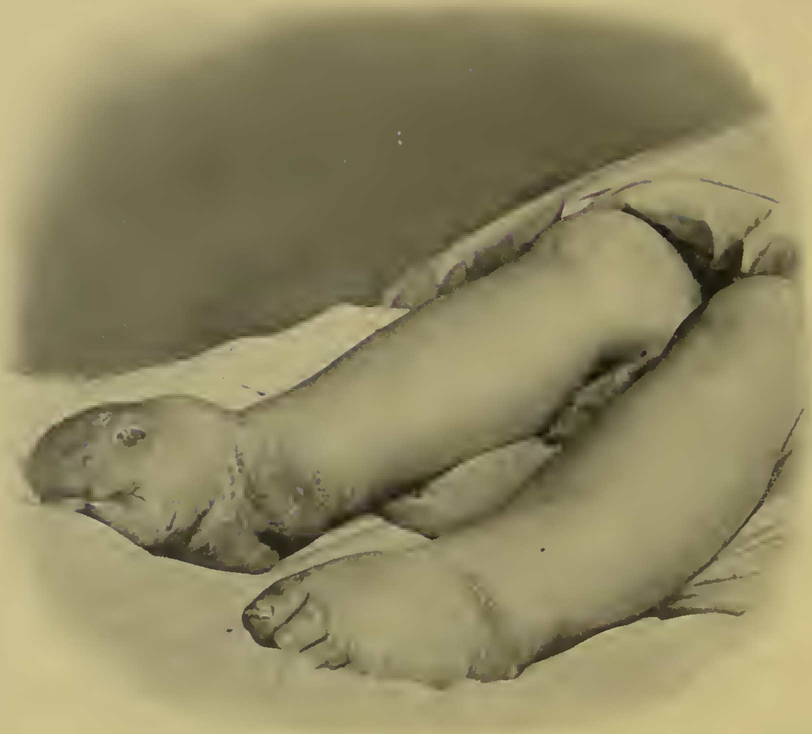


Fig. A.—Represents the condition before the administration of thyroid gland. Note the œdema, also the presence of an ulcer on the dorsum of the right foot.

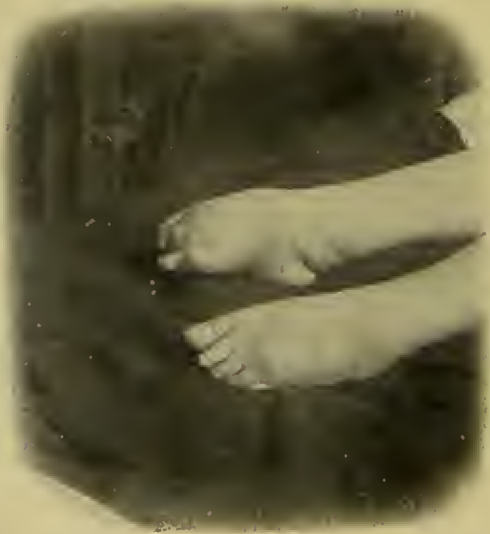


Fig. B. Shows the diminution of swelling and the healing of the ulcer following thyroid medication.

(By kind permission of Dr. Macalister).

previously had developed rheumatoid arthritis. The disease had involved not only the joints of the hands and fingers but also the large articulations of the limbs and those of the cervical spine. In addition, intensely painful nodules developed from time to time on the aponeurosis of the occipito-frontalis. The joint swellings were of the usual peri-articular character, with apparently no bony involvement.

Shortly after admission to the Home for Incurables under Dr. Macalister's care, she sustained a cerebral hæmorrhage, which was followed by right-sided hemiplegia and temporary loss of speech. The aphasia did not last long, and the arm and leg, after remaining helpless for weeks, recovered some degree of power.

Following this apoplectic attack, however, gradual swelling of her limbs and trunk developed, especially of the lower extremities, which became greatly enlarged, the calves measuring 22 in. in circumference.

There was present no cardiac or pulmonary condition which could explain its incidence, and the urine contained no albumin.

Mentally she was extremely depressed, frequently wept, and betrayed no interest in her surroundings.

Her condition now became complicated by the appearance on the dorsum of her right foot of an ulcer, which was deep, round in shape, and of a punched-out character.

It persisted for months and had proved intractable to all forms of treatment, when the similarity of its lining surface to mucoid tissue suggested that it might be of a myxœdematous nature.

Thyroid gland was accordingly administered in gradually increasing doses up to 15 grams daily. Under this treatment, not only did the ulcer heal, but the swelling of arms, trunk, and legs disappeared, so that within three months the calves measured only 14 in.

The mental depression vanished, and in addition the joints improved, becoming reduced in size, less painful, and more mobile.

This was especially noticeable in the neck, which, formerly stiff, painful, and creaking, became painless and movable. For the accompanying illustrations (*Plate XI*) showing the condition before and after the administration of thyroid, I am much indebted to Dr. Macalister.

Paralysis agitans, as Charcot noted, may supervene in the subjects of rheumatoid arthritis, and in view of Buzzard's statement that some obscure pathological connection appears to exist between the two conditions, it is worthy of note that quite a few instances have been noted of the co-existence of the two diseases.

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CHAPTER IX.

DIAGNOSIS AND PROGNOSIS.

DIAGNOSIS.

THE diagnosis of the various forms of rheumatoid arthritis is best dealt with in two sections, the first pertaining to the more acute forms ; the second to those of a more chronic nature. The distinctive features of the more acute forms may be summarized as follows :—

GENERAL DIAGNOSIS.

It is a polyarticular affection, occurring most commonly in young women. Its onset is more or less acute. It begins in the small joints, spreading centripetally, with a tendency to symmetry. The disease does not flit from joint to joint, the joint swellings being persistent, and nearly always leaving some residual thickening. The skin over the affected joints is semi-asphyxial in tint, or unnaturally white. Local pain and tenderness are slight or absent. The swelling is due to peri-articular thickening more than effusion. The temporo-maxillary and cervical articulations are almost always involved. Rapidly induced wasting of interossei and extensor groups of muscles ensues, with increased reflexes, while glandular enlargements are not infrequent. Pigmentation, vasomotor troubles, localized as well as generalized sweats, are additional features. Cardiac troubles are very rare. The temperature is raised, but low compared with other arthritic diseases, especially considering the widespread affection of joints. The slight, though continuous, pyrexia, a quickened pulse-rate, and the persistent nature of joint swellings, taken together, are very suggestive of rheumatoid arthritis.

DIFFERENTIAL DIAGNOSIS.

The diseases most likely to be confounded with acute rheumatoid arthritis are acute rheumatism, acute gonorrhœal rheumatism, acute gout, etc.

From Acute Rheumatism.—Taking all ages collectively, acute rheumatism is more common in males, acute rheumatoid in females. Acute rheumatism begins in the large joints in preference to those of the hands and fingers. It shows a marked tendency to flit from joint to joint. The affected joints recover completely, leaving no residual thickening. The skin over the affected joints usually shows a distinct red blush. The joints are intensely painful and tender to the touch. The swelling is due to synovial effusion. The temporo-maxillary and cervical articulations are very rarely involved. Wasting of muscles is rarely, if ever, present, and never ensues with such rapidity. The temperature is, as a rule, higher than in acute rheumatoid arthritis. Profuse general sweats are more characteristic than localized. Cardiac complications are common. A quickened pulse-rate is, as a rule, not so noticeable a feature as in rheumatoid arthritis, where cardiac lesions are very rare. There is an absence of vasomotor, pigmentary, and trophic disturbances; and treatment with salicylates is, as a rule, successful.

From Acute Gonorrhœal Rheumatism.—In this disease the presence or history of a urethral or vaginal discharge will afford a valuable, though in the absence of positive bacteriological results not an infallible, aid to the diagnosis of the true nature of the joint affection. The absence of marked fever; the limitation of the affection to the larger joints, especially the knees; the special involvement of the fasciæ, notably the plantar; the occurrence of iritis and conjunctivitis—all these are points in favour of gonorrhœal rheumatism.

Special difficulty arises when a polyarthritic condition, indistinguishable from rheumatoid arthritis, follows gonorrhœal infection. In such cases the following peculiarities are suggestive of its being of this nature: involvement of the sterno-clavicular joint; marked implication of the vertebral articulations, in addition to the polyarthritis of the extremities; the presence of iritis and conjunctivitis. In the absence of acute ocular manifestations I have found it of value to note the contour of the pupil, whether irregular or not. In the case of the former the instillation of atropine may reveal the presence of synechiæ or pigmentary deposits on the lens, evidences of past iritis.

From Acute Gout.—Confusion is hardly possible, save in the rare event of an acute polyarthritis of gouty nature. Here the age, habits, and previous history of an attack limited to the great toe, coupled with the presence of tophi, and the results of uric acid estimation, will hardly fail to indicate the correct diagnosis.

From Secondary Syphilitic Arthritis.—It is just possible that confusion may arise in a case of polyarthritis of this nature when accompanied by prolonged pyrexia. The previous history, the presence of secondary eruptions, and nodes on the shafts of the long bones, are the points on which to rely. Recently two cases of this nature came under my care, and promptly responded to antisyphilitic treatment.

With the exception of acute gout, it will be noted that all the foregoing joint affections with which rheumatoid arthritis of the more acute type is likely to be confounded belong to that large group known as the infective arthritides. Now, in all the diseases hitherto referred to in this connexion, their association and dependence upon a specific micro-organism has been clearly established, if we except the scepticism with which some still view the relationship of the *Micrococcus rheumaticus* to acute rheumatism. Apart from these, however, there still remain a number of joint affections hitherto unaffiliated to any special germ, and with which, undoubtedly, acute rheumatoid arthritis is not infrequently confounded. Now, broadly speaking, these cases of infectious arthritis are divisible into two groups—suppurative and non-suppurative—the more destructive character of the former being due to the actual presence of pyogenic organisms within the joint cavity, while the arthritis in the latter is presumably due to the action of toxins derived from some remote source of infection.

Further consideration of the suppurative varieties need not detain us, as all authorities agree that in the rare cases in which pus formation has been noted in rheumatoid arthritis it was undoubtedly due to secondary infection.

It now remains for us, as far as possible, to indicate those clinical features which distinguish acute rheumatoid arthritis from those non-purulent types of infective arthritis unassociated with any specific germ. In the lack of positive bacteriological evidence in either case we are compelled to rely upon certain clinical characteristics which may effect a cleavage.

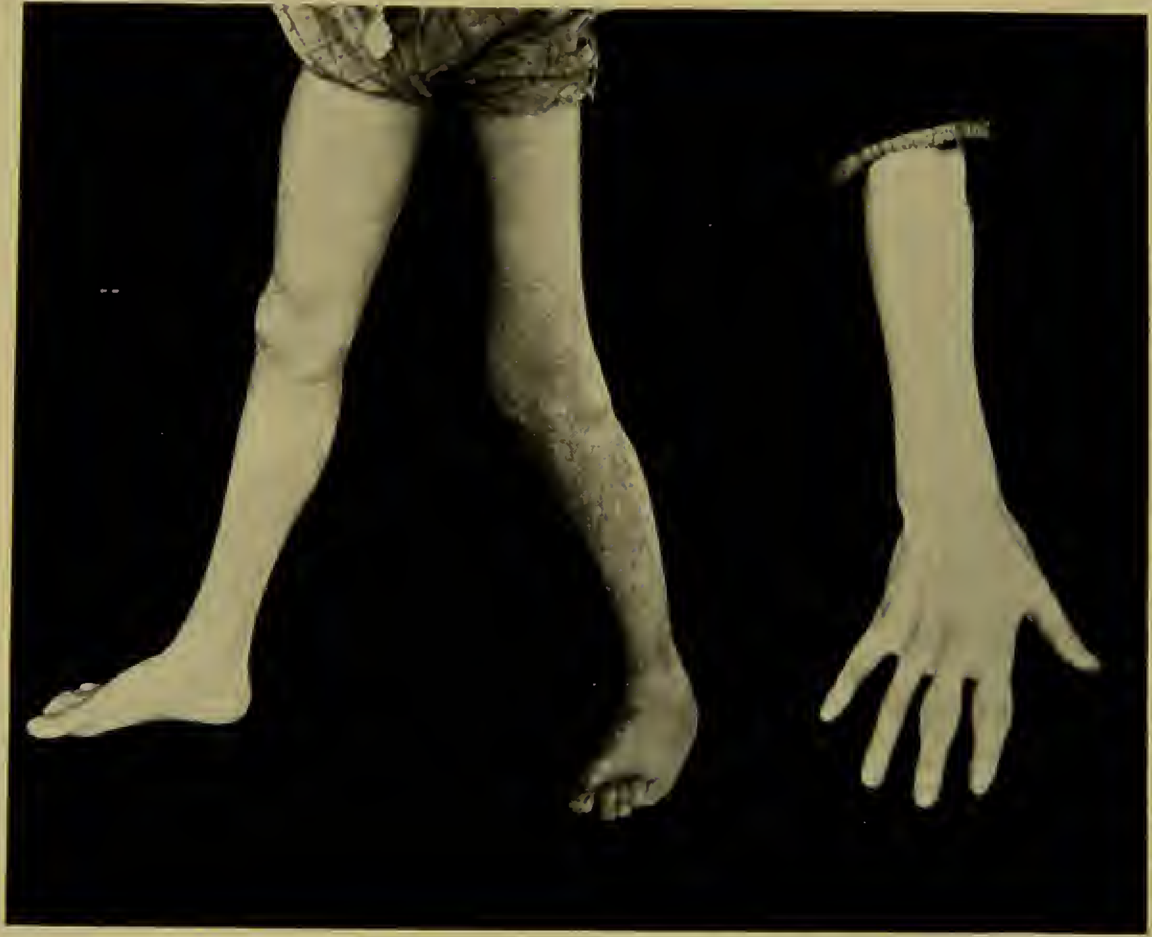
Unfortunately, no help is afforded us by a consideration of the age incidence of the two varieties of joint disorder, as both have been observed at all periods of life ; but something may be gathered from the very marked predilection of rheumatoid arthritis for the female sex. The previous history, if it reveal the recent occurrence of some infective malady, known to be followed occasionally by non-purulent arthritis due to the same microbic agency, such as dysentery, typhoid fever, pneumonia, etc., will be of value, and will constitute strong presumptive evidence that we are dealing with an infectious arthritis following one or other of these diseases.

Similar inferences may be drawn from the presence of any local source of infection, such as pyorrhœa alveolaris, otorrhœa, and so forth.

Where such is not forthcoming, reliance must be placed on the following characteristics, which appear to distinguish cases of infectious arthritis in general from rheumatoid arthritis. Thus, for example, in infectious arthritis the general symptoms are often much more acute, the temperature is of higher grade, enlargement of the lymphatic glands in association with the affected joints is almost invariable, the spleen is also more commonly enlarged, while leucocytosis and a secondary anæmia are usually present.

Passing to the articular lesions, though all, or nearly all, the joints may be involved in any given case of infectious arthritis, yet such widespread invasion is by no means the rule. What happens in the majority of instances is that at the onset of the malady the disease attacks one, two, or three joints, and having done so, it shows no tendency to involve any other joints, even after weeks or months have elapsed, during which time the body temperature may be more or less continuously raised, and of a septic or irregular type. Again, the mode of distribution of the arthritic lesions differs in the two. In infectious arthritis (*Plate XII*) the larger joints are by preference attacked, though I have seen exceptions to this rule in the case of the metacarpo-phalangeal and proximal phalangeal joints. As opposed to this, rheumatoid arthritis almost invariably begins in the small joints

PLATE XII.



Infectious Arthritis of Left Knee, with marked atrophy of thigh muscles of same limb.
Hand of same patient, showing spindle enlargement of mid-phalangeal joints of index and middle fingers

of the hands and fingers. Moreover, in the former the distribution of the lesions is erratic, while the tendency in rheumatoid arthritis is towards symmetrical involvement of the joints.

With regard to the appearance of the joint swellings one often meets or hears of the phrase, " typical fusiform or spindle-shaped joint " of rheumatoid arthritis, as if it were peculiar to this disease: whereas, as Goldthwait points out—and my own experience confirms him—such is not the case: equally typical examples of spindle-formed joints are to be met with in infectious arthritis. This is not to be wondered at, inasmuch as this peculiar shape is dependent upon the structures involved, and, owing to their exposed situation, is more noticeable in the phalangeal joints. Again, the joint the seat of infectious arthritis presents a definite red blush in most cases, whereas in rheumatoid arthritis it is unnaturally white or bluish in colour. Wide-spread inflammatory œdema of the affected limb is more common in infectious arthritis.

Seeking for further distinctive features, other than those peculiar to the arthritic lesions, we find in acute rheumatoid arthritis, as opposed to infectious arthritis, an abundance of collateral phenomena. In those rare instances in which rheumatoid arthritis remains for some time confined to the small articulations of one hand (enhancing the possibility of confusion with infectious arthritis by this asymmetrical distribution), the presence of vasomotor phenomena, neuralgic pains, or muscular cramps in the opposite hand will be in favour of its being rheumatoidal in nature.

In order to further emphasize the special features of infectious arthritis, I shall cite in brief the clinical history of one or two cases :—

A. B., male, aged 30, was seized suddenly with severe pain in the right forearm radiating down the dorsal surface from the elbow to the fingers, quickly followed by intense swelling of the forearm and hand ; simultaneously he was attacked by severe pain along the back of the left thigh, and tenderness and swelling of the dorsum of the foot of the same side ; in addition, there was considerable constitutional disturbance, the temperature rising to 103° F. Either as the result of treatment by aspirin, or spontaneously, the temperature and the swelling in both affected regions rapidly subsided, when it was seen that the metacarpo-phalangeal joints of both thumb and index finger of the right side were enlarged, tender, and slightly reddened, likewise the left ankle. On further examination he was found to have purulent right-sided otorrhœa of many years' standing; and, in addition, a dental plate concealed a number of carious stumps. I came to the conclusion that it was a case of infectious arthritis, due, probably, to absorption from these two foci of suppuration, to the removal of which I directed my treatment. Accordingly, the carious stumps were removed, and antiseptic treatment was directed to the ear. As the result of these measures, the enlarged joints rapidly diminished in size, all pain disappeared, no extension to other joints took place, and a year afterwards, when I saw him again, he had had no relapse, and his joints, though slightly thickened, gave him no further trouble.

Female, single, aged 25, native of the Transvaal, while on a visit to England, after a variable period of malaise of indefinite nature, developed acute pain and swelling in the left ankle joint, attended by œdema of the whole leg, extending half-way up the thigh. The case was thought to be one of acute rheumatism complicated by phlebitis. Shortly after, one of the right metacarpo-phalangeal joints became painful and swollen, but no further extension to other joints took place, notwithstanding that the temperature, high at onset, remained continuously above normal for six or seven weeks, with considerable diurnal range. No cardiac complications were present throughout the illness. As soon as feasible, she was sent to Bath for treatment. I could discover no possible focus of infection, with the exception of a previous history of having suffered from what was termed "malarial typhoid." I may add that she was the subject of long-standing and extensive pustular acne, and it is possible that infection took place from this source. As regards treatment: I placed her on a generous diet; treated the acne with sulphur and resorcin; and, as soon as her temperature subsided, gave her a course of Aix-massage baths, paying special attention to the affected joints. This, coupled with passive resistance movements and exercises, removed the œdema, and restored the mobility of the joints perfectly. Six months afterwards I heard that she was able to follow the outdoor pursuits of an active colonial life without the least difficulty.

Female, aged 24; married; two children. Six months previously was suddenly seized with pain and swelling in right ankle joint. The temperature became raised, and after a few days pain and swelling ensued in left knee joint, the affected ankle in the meantime having spontaneously recovered. The knee joint became intensely swollen, with a slight redness of the overlying skin, and it was feared that suppuration was about to occur. The limb was placed at rest upon a splint, in order to avoid a faulty position in the event of ankylosis ensuing. The pyrexia continued for some weeks, and was, with the arthritic condition, quite refractory to treatment with salicylates. No cardiac lesions were present throughout the illness, and saving the slight initial involvement of the right ankle, the condition was strictly limited to the left knee. After some weeks the inflammatory condition in the knee quieted down, leaving the knee permanently fixed in extension. X-ray examination disclosed no bony changes, the immobility being apparently due to fibrous adhesions. There was no history of gonorrhœa, and, apparently, no other localized source of septic absorption.

Male, aged 33; married. As a result of a sprain some fifteen years previous had suffered from weakness of the left knee. From time to time, especially after undue exertion, had been liable to pain and recurring effusions, which led to a diagnosis of a loose semilunar cartilage. When he came under my observation the knee-joint showed signs of chronic thickening of the synovial and peri-articular structures, with slight creaking on movement. There was no clear history of any previous infection; but on examination a number of carious stumps were found underneath a dental plate in the upper jaw. Accordingly I advised removal of these stumps as a possible source of infection. Subsequently, the irritability of the joint declined, and he was able to take athletic exercise with impunity.

In the foregoing cases it will be noted that the joint lesions were limited in distribution to only a few articulations. Moreover, they showed no tendency to progressively involve joint after joint; even when, as in the second and third examples, the pyrexia of high grade was maintained for a considerable period.

Had they been instances of acute rheumatoid arthritis, the disease would have by that time implicated all, or a large number, of the articulations.

Again, the distribution of the diseases was erratic, displaying no tendency to symmetry. Two of them were accompanied by a marked degree of inflammatory œdema of very painful character.

In none of them was there present any noteworthy degree of muscular atrophy : an important distinction between them and acute rheumatoid arthritis.

While these, together with certain differences before alluded to, constitute the chief points of distinction, it must not be forgotten that the dividing line between these infective arthritides and the more acute forms of rheumatoid arthritis is by no means sharply defined. In all probability a satisfactory means of differential diagnosis will not be available on purely clinical grounds, but on bacteriological data, which it is to be hoped will before long be forthcoming.

The more chronic forms of rheumatoid arthritis require to be differentiated from osteo-arthritis and chronic gout. For the purpose of greater lucidity and avoidance of repetition, it seems wiser to defer the discussion of this question to a later chapter, when the clinical account of osteo-arthritis will have been perused.

We will therefore pass on to describe the X-ray appearances of infectious arthritis, rheumatoid arthritis, and osteo-arthritis, together with chronic gout (*Plates XIII*, *XIV*, *XV*).

Much importance of late years has been attached to skiagraphy as an aid in the differential diagnosis of joint diseases. Among the first to lay stress upon its importance in the diagnosis of the various forms of arthritis deformans was Goldthwait, of Boston. According to this authority no changes can be detected by skiagraphy in the bone or cartilage in infectious arthritis, the density of the former being in no way lessened, while the cartilage is of normal thickness. If, however, the infectious arthritis is of a destructive character, new bone may be thrown out in the process of repair ; and if, in such cases, sole reliance be

PLATE XIII.

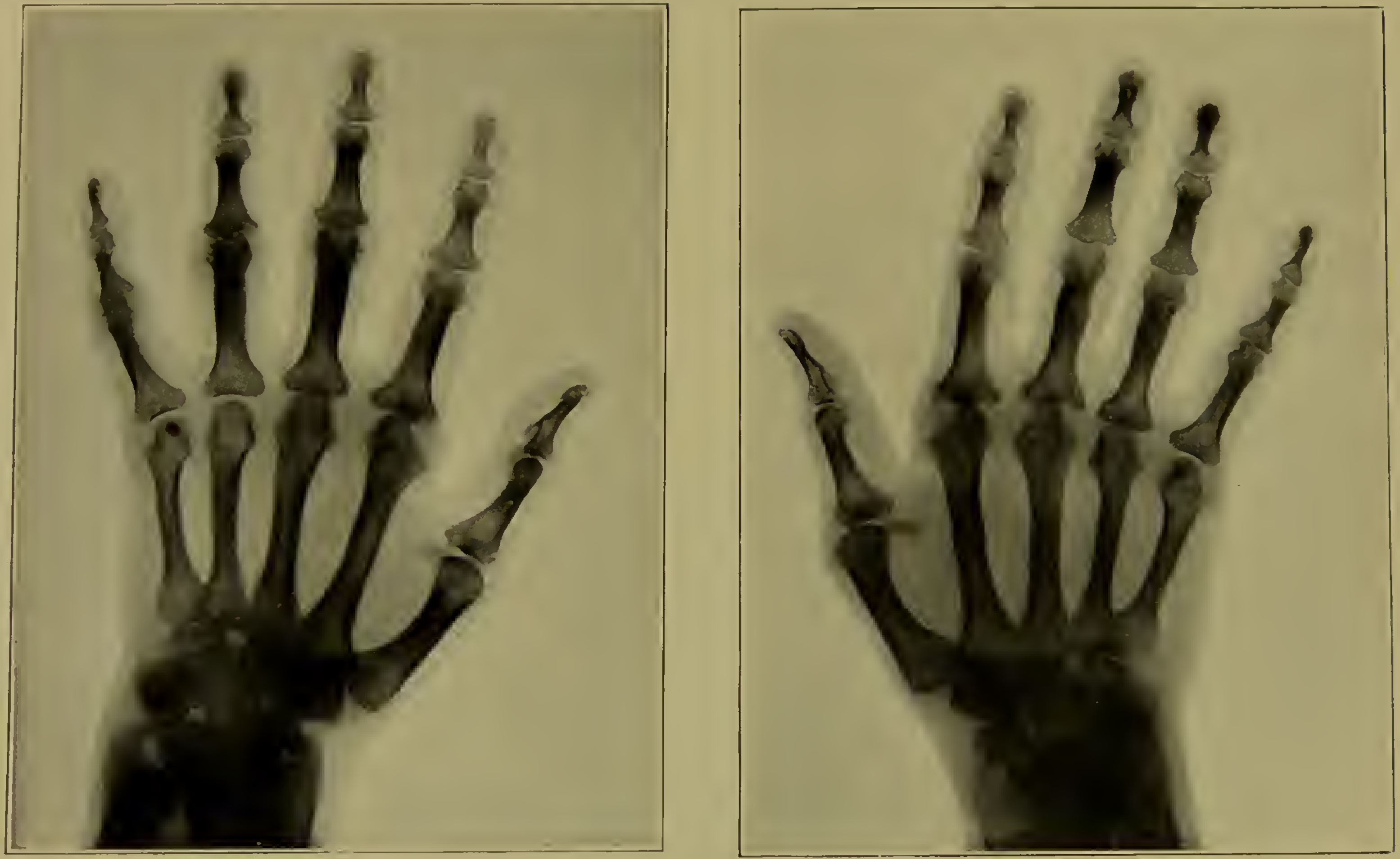
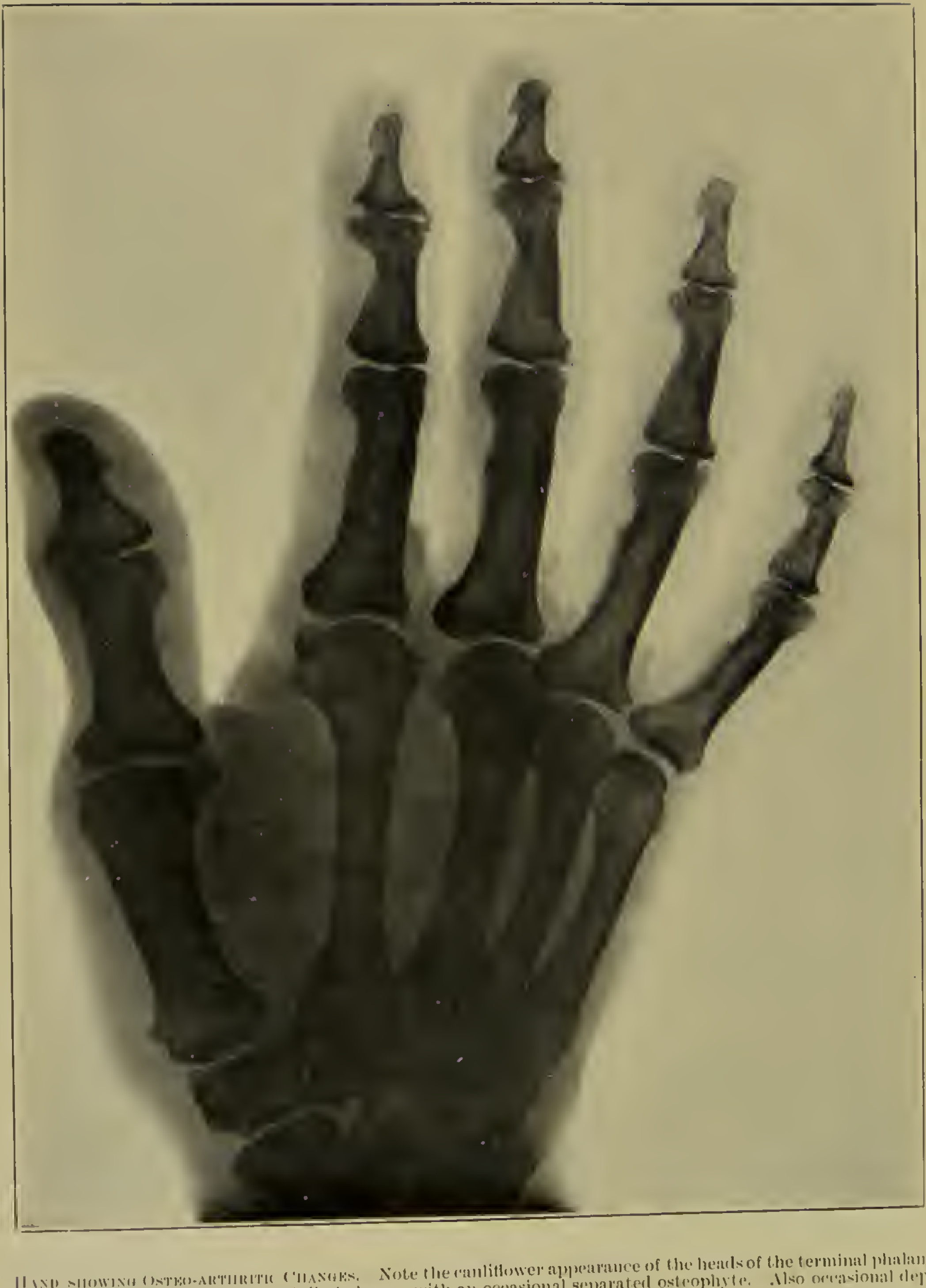


Fig. A. *Fig. B.*

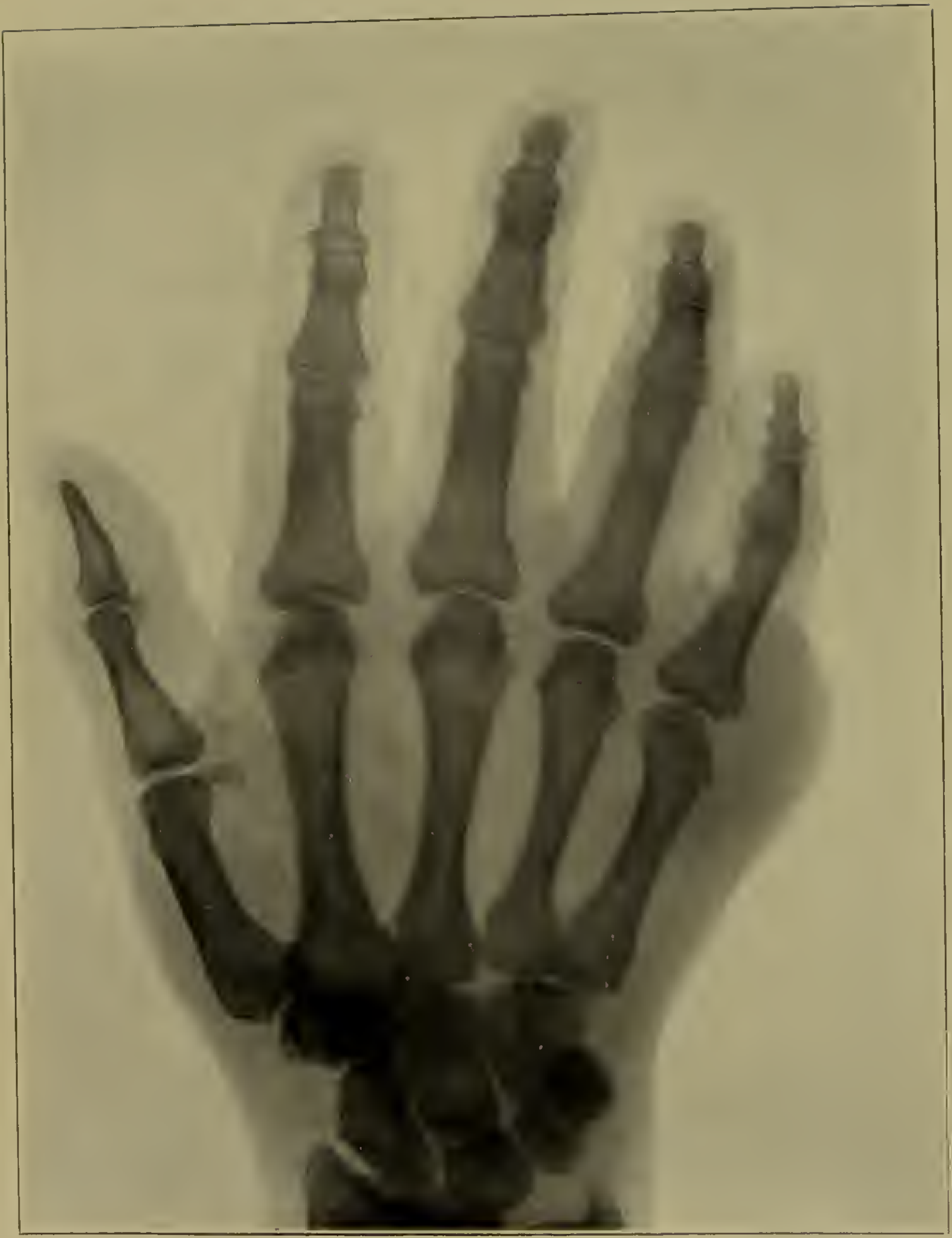
Skiagraph of Chronic Rheumatoid Arthritis.—Atrophy of cartilage of metacarpo-phalangeal joints of index and middle fingers, with destruction of bony tissue of heads of metacarpal bones, in both hands. *Fig. A* shows atrophy of cartilage in mid-phalangeal joints of little, ring, and middle fingers.

PLATE XIV.



Hand showing Osteo-arthritic Changes. Note the cauliflower appearance of the heads of the terminal phalanges, also deposits "frilling" at the bases of all phalanges with an occasional separated osteophyte. Also occasional deposit on the middle of the shafts.

PLATE XV.



SKIAGRAPH OF CHRONIC GOUT IN A MAN.—Shows a large swelling in the soft tissues on the ulnar side of the hand, due to gouty deposit.

placed on X-ray appearances without any reference to the clinical history and course of the disease, then, as he says, this irregular formation of bone is likely to be confused with the X-ray appearances of osteo-arthritis. It is, however, possible, according to him, to distinguish by careful scrutiny of the skiagraphs the nature of the case. The new formation of bone in these destructive forms of arthritis conforms in every way to that seen as a result of septic osteitis or periostitis, the osteophytic outgrowths taking place at the point of infection, wherever that is present, and not, as in osteo-arthritis, at the margin of the cartilage.

In *rheumatoid arthritis* absorption of the cartilage takes place to a variable degree; in some cases it is completely absent. According to Goldthwait, this change may be observed in the very earliest stages. The bones participate in the pathological change, the first evidence of which is an abnormal translucency to the rays, usually, but not always, confined to that portion of the shaft entering into the joint. Eventually, the articular ends of the phalangeal and metacarpal bones may undergo erosion, in some cases to a very marked degree, the bones more or less telescoping into each other.

In *osteo-arthritis* deposits of new bone at the margins of the cartilages can be detected, and with the advance of the disease the shafts of the bones in their neighbourhood become more dense. According to Ironside Bruce, in early cases the head of the bone may show a greater translucency than is normal, the translucent area being sharply marked off from the neighbouring bone, which is of normal density.

In chronic gout gouty deposits can be discerned by skiagraphy, giving rise to a noticeable shadow. The fingers or toes are the best sites in which to make out these deposits.

Owing to the fact that considerable disintegration of the bone may result, the appearances in some instances resemble those found in skiagraphs of rheumatoid joints. Stress has been laid on this similarity by Strangeways, of Cambridge, and it was also previously remarked by Goldthwait, who, however, claims that while in atrophic or rheumatoid arthritis the bone, though thin, maintains its outline, on the other hand, in gout, the outline of the bone is not distinguishable, owing to the more complete destruction of the bone by the morbid process. This observer states that the resemblance to rheumatoid arthritis is most likely to obtain when the articular ends of the bones are the seat of the disease. In other instances, however, the shafts of the bones may be affected by the gouty diseases, in which case they show punched-out areas simulating syphilitic dactylitis.

Recent researches have shown that the morbid process in rheumatoid arthritis may not invariably be wholly atrophic, but is, in rare instances, accompanied by deposits of new bone, as evidenced by skiagraphy. This fact, taken in conjunction with the striking resemblance between the skiagrams of chronic gout and rheumatoid arthritis, seems to indicate that it is unwise to rely wholly upon skiagraphy for our differential diagnosis of these conditions.

Skiagraphy, though of great and probably increasing value, cannot, for one moment, be allowed to take the place of careful observation of the general clinical features of our cases, to which it must always be considered subsidiary.

PROGNOSIS.

The prognosis of rheumatoid arthritis will naturally vary according as to whether we attempt to gauge the outlook in the early and active stage of the disease, when it is, so to speak, in the making, or whether we regard it in its later stages, a period of its life-history when, strictly speaking, we are not dealing with rheumatoid arthritis, but with its malign, and for the most part irremediable, results.

In the former period of its course much will depend on the promptness with which the true nature of the malady is recognized. This may seem a trite observation, but I think that no one will dispute that all of us, at one time or another, have felt the difficulty of swiftly arriving at an accurate diagnosis in the more acute forms which so closely resemble acute rheumatism, etc. Doubtless, in many instances, much of the delay is due to the fact that we are too apt to forget that there is such a condition as acute rheumatoid arthritis; and the consequences of treatment conducted along more or less depressing lines under a misapprehension undoubtedly darken the outlook. If, however, a timely recognition of its true nature be achieved, the prognosis is, I think, reasonably good; and, although it may be beyond the power of therapeutics to restore the more or less disorganized joint to its pristine state of integrity, it is, nevertheless, possible to arrest the progress of the disease, or its extension to other articulations. As I have before stated in the text, I am of opinion that, especially in those cases whose course is characterized by a series of exacerbations and remissions, the persistence in the intervals of an accelerated pulse-rate, the continuance of vasomotor phenomena and sweating, with the maintenance of an increased myotatic irritability, are features of grave omen, evidences of the continuous character of the underlying toxæmia, and indicative by their presence of an oncoming exacerbation of the malady. Other factors that also enter into the question are those relating to the patient's position in life : his powers, or not, of obtaining the generous nourishment such cases need, his chances of exposure to extremes of temperature, and damp, and especially his liability to mental worry and care : all of which environmental circumstances, if not favourable, undoubtedly promote the steady progress of the disease.

The unrelieved gloom hitherto overshadowing the secondary or terminal stages of the malady has in some measure been dissipated by recent advances in the surgical treatment of the deformities.

The striking success that has followed surgical intervention in some instances materially brightens the outlook of these unhappy beings, whose lives, when bedridden, can only be characterized as one long-drawn-out misery.

Chapter X.

TREATMENT.

GENERAL CONSIDERATIONS.

Recognizing that rheumatoid arthritis is a progressive, wasting disease, one characterized by atrophy, not only of bone and cartilage, but also of the skin and subcutaneous and muscular structures, it is clear that a depletory mode of treatment is, with few exceptions, carefully to be avoided.

It should never be forgotten that the articular lesions in this affection are only one feature of a very complex clinical syndrome. This, in itself, indicates that any course of treatment which does not include attention to the general health of the patient is foredoomed to failure.

Consequently, our primary object should be the correction of any abnormal conditions which react injuriously upon the well-being of the organism.

Inasmuch as the pathological changes before alluded to are now universally considered to be toxic in origin, it follows that this view of the pathogeny carries with it the inference that a most careful search should be instituted for the detection in the patient of any localized source of toxic absorption.

LOCALIZED FOCI OF TOXIC ABSORPTION.

The sites in which such are most likely to be met with are the mouth and its accessory cavities.

A condition of oral sepsis may sometimes be detected, occasionally one of pyorrhœa alveolaris, while in other instances the presence of a chronic nasal or aural discharge, purulent or non-purulent, may be ascertained.

Too much stress cannot be laid upon a thorough examination of the mouth and nasopharynx. Carious teeth, when present, should either be thoroughly cleansed and stopped, or else extracted. Unhealthy conditions of the gums must also be attended to. For this purpose, the daily use of antiseptic mouth-washes containing peroxide of hydrogen, supplemented by the intelligent and thorough use of the tooth-brush, should be insisted upon.

As a rule, in patients of the better class, such conditions have been attended to ; but in all persons wearing dental plates the statement usually volunteered, " that all the stumps have been removed," must not be accepted too readily. Only too frequently, on examination, one finds that the decayed stumps, instead of being drawn, were merely broken off or filed down, and the fangs, often suppurating, remain behind in their sockets. The result is, that the foul discharge is pent up behind the plate, from which it escapes at intervals, to infect the buccal secretions and food.

Where such a condition of oral sepsis exists, the extraction of the stumps must be insisted upon as the basis of successful treatment.

Only by the effectual use of such measures is it possible to prevent the constant infection and reinfection of the lower levels of the alimentary tract by putrefactive anaerobes swallowed automatically with the food.

The next step consists in thorough examination of the tonsils. When we consider that the advisability of removing these glands, even during the course of acute rheumatism, has been seriously suggested, we at once appreciate the increasing importance attached to these structures as sites of infection. In any case of rheumatoid arthritis in which recurring attacks of tonsillitis have been followed by exacerbations of the joint condition, it would seem justifiable to advocate their excision as a prophylactic measure against future attacks.

Similarly, the nasal passages, with their accessory cavities—the antral and ethmoidal sinuses—should be examined. Because of the difficulty in excluding disease in these regions, I think the services of an expert should be enlisted more frequently than is at present usual. Recently, I saw a young woman, the subject of rheumatoid arthritis in an early stage, affecting the small joints of both hands. The patient had for some years previously suffered from an intermittent sanious discharge from the left nostril. As a result of local treatment, the nasal condition soon got well, and was quickly followed by an amelioration of her joint troubles, from which she subsequently completely recovered.

Macalister,[1] of Liverpool, and others, have emphasized the importance in rheumatoid arthritis of not overlooking this possibility of toxic absorption from the nasal passages.

Aural discharges of long standing and purulent in character are also to be met with occasionally, and likewise demand special local treatment.

The genito-urinary tracts, both in the male and female, must be examined from the point of view of their being possibly a source of toxic absorption. Gonorrhœa, we know, can produce a polyarthritic condition clinically indistinguishable from rheumatoid arthritis. Consequently, this etiological factor, especially in the case of males, should not be excluded too readily. When a history, even though remote, of such venereal trouble is forthcoming, the absence of discharge does not preclude this as a possible *fons et origo mali.* Bloodgood,[2] dealing with this question of the etiology of the chronic arthritides, warns us that frequent massage of the prostate may be necessary before the presence of the gonococcus can be demonstrated; hence the difficulty of exclusion.

The same author refers to the possibility that, in similar cases, the endometrium in women may be the site of infection by the gonococcus and other organisms.

West[3] has reported an instance in which treatment of an inflammatory condition of the bladder was followed by cure of the apparently related rheumatoid arthritis.

In the same manner, encouraging degrees of improvement in the arthritic condition have followed treatment of other local conditions, when present, such as ulcerated piles and uterine disorders.

While detailed examination along the lines indicated constitutes the primary step in treatment, it is obvious that the degree of success that may attend such procedures will vary according to the period of the malady at which they are undertaken. If definite foci are detected and promptly treated during the early stages of rheumatoid arthritis, the happiest results may be achieved.

Here, again, I would emphasize the importance of the premonitory symptoms before alluded to, whose occurrence in patients suffering from such localized affections is so suggestive that some degree of toxic absorption is taking place from these sites.

Unfortunately, in all too many instances of rheumatoid arthritis there are no such definite indications as to the source of absorption. Consequently, we have often to rely upon indirect means to combat the results of a toxæmia whose origin is cryptic. These will be discussed under various headings: climatic, dietetic, and so forth.

CLIMATE.

Most authorities agree that the conditions to be sought for are those of dryness and warmth.

In other words, a low relative humidity, abundant sunshine, and a small rainfall, constitute the ideal climatic surroundings for rheumatoid patients.

Undoubtedly, the disease is aggravated by damp; and sudden fluctuations in temperature are also apt to be followed by the same untoward results.

Residence at the seaside, too, has the effect of increasing the painfulness and stiffness of joints.

Unquestionably, the most desirable sites for wintering in are those of Upper Egypt and Algiers, where we get the advantages before alluded to, to a great extent; the only drawbacks to residence at these spots being the wide diurnal range of temperature, and the not infrequent presence of cold winds.

These latter, however, are not so prevalent as in the French Riviera, in which region, according to Weber,[4] the neighbourhood of Grasse seems the most suitable for persons suffering from this affection.

Unfortunately, the length of the journey makes it undesirable for patients in an advanced stage of crippledom to attempt it, not to mention the fact that financial considerations debar many from entertaining any such project for their amelioration.

Those that, either by choice or from necessity, remain in the British Isles, usually find that an inland climate suits them best; and for this reason Bath, enjoying as it does a comparatively large amount of sunshine with a low diurnal range of temperature, is deservedly resorted to by this class of patients.

DIET.

In the section devoted to etiology, we emphasized the probability that the fundamental condition in rheumatoid arthritis is one of autotoxæmia ; moreover, that the significance of the oral and naso-pharyngeal sepsis lay in the fact that they either initiated or promoted the continuance of the gastro-intestinal disturbances so frequently observed in this condition.

The toxæmia resulting from the abnormal putrefactive and fermentative processes associated with these conditions, indicates that great care should be exercised in dieting these patients. Indeed, as recent researches have shown, in the judicious selection or variation of diet lies our most effectual means of controlling the growth, or modifying the character, of the intestinal flora responsible for the initiation of abnormal putrefactive and fermentative processes, with their remote consequences—chronic toxæmia.

During the *acute* stages of the disease the nourishment taken should be largely fluid in kind, and such as admits of easy digestion and rapid absorption by a febrile patient. Under such circumstances milk will form the staple food.

The amount taken will vary with the individual's capacity for digesting it. While some people find no difficulty in assimilating quantities varying from 2½ to 3 pints or more in the twenty-four hours, others soon experience nausea, vomiting, and even diarrhœa, from the passage of undigested curds. It is therefore advisable to begin with small amounts given at regular intervals.

Where dyspeptic symptoms supervene, these may be minimized by diluting the milk with equal quantities or more of some effervescent alkaline water, or by the addition of three to five grains of citrate of soda to each ounce of milk.

Where milk agrees, it should be given up to the limit of the patient's digestive capacity, and its nutritious qualities may be enhanced by the yolk of an egg beaten up in it.

Again, in some instances, patients who object to plain milk will readily partake of one of the prepared foods—Benger's or

Mellin's—made up with this vehicle. Plasmon and Sanatogen also deserve mention in the same category.

According to Albu,[5] a milk diet constitutes the most effectual means of attaining a comparative degree of intestinal antisepsis.

This antiputrefactive effect of milk is attributable to the lactose it contains, which, under the action of the bacteria of the small intestine, is decomposed into lactic and succinic acids (Blumenthal). These acids inhibit the growth of the proteolytic anaerobes, which cause putrefaction.

These considerations are of great importance, when we take into account the frequency with which excessive intestinal putrefaction has been observed in rheumatoid arthritis, especially in view of the constancy with which excess of indican and ethereal sulphates in the urine was noted by Herter in early cases of the affection.

The knowledge that lactic acid bacilli reduced intestinal putrefaction, suggested the enhancing of this beneficial action by resorting to a diet consisting largely of curdled or soured milks. Thus, Andrews and Hoke[6] record five cases of rheumatoid arthritis attended by excessive intestinal putrefaction, in whom treatment by fermented milk was followed by marked amelioration both of the local and general conditions. Cows' milk fermented by the action of lactic acid bacillus and brewers' yeast was used. In some instances it was the sole article of diet, as much as three to four quarts daily being ingested.

Tubby,[7] from his own experience, strongly advocates the adoption of this diet in the acute polyarticular form so common in young women, as well as in the more chronic types.

Before placing the patient on a strict regimen of soured milk, the presence of an excess of indican and aromatic sulphates in the urine should be established.

As Herschell[8] remarks in his admirable *résumé* on the use of lactic acid ferments in the treatment of disease, it is absolutely necessary that the strictest ritual should be observed in the preparation of the soured milk.

The natural ferments, the Bulgarian maya and the Egyptian leben, are open to the objection that they contain not only lactic acid bacilli, but other bacteria also, as well as yeasts.

The tendency nowadays is to employ pure cultures of lactic acid bacilli. Opinions differ, however, as to whether liquid cultures in tubes or dried cultures in the form of tablets or powders should be utilized for the preparation of the soured milk. Herschell, for example, prefers the latter as more reliable, and in the few instances in which I have adopted this mode of treatment my experience accords with his as to the superiority of the dried cultures.

The somewhat detailed consideration given to this method of treatment is, I think, fully justified in view of the notorious difficulty that attends the treatment of these acute forms. While, reasoning by analogy, the success that has attended this form of treatment in all varieties of auto-intoxication strongly indicates its further employment in rheumatoid arthritis, still the limited number of cases hitherto recorded forbids any dogmatic statements as to its therapeutic value.

Where there exists an insuperable aversion to milk in every form, or a very limited power of assimilating it, there need be no hesitation in supplementing, or even replacing it, by a liberal allowance of farinaceous foods. Thus, carefully prepared thin oatmeal, or barley gruel, will often afford a pleasant change, the palatability of which may be increased by the addition of salt or grape sugar, flavoured with nutmeg, lemon-peel, or other agreeable spices. Arrowroot, too, is serviceable, and, like the former, may be prepared with or without milk. Milk puddings, such as rice, sago, tapioca, etc., to which an ounce of ground malt or a tablespoonful of maltine has been added, are often acceptable to the patient, varied with custards and blancmanges made with gelatine.

When, as not infrequently happens in these distressing cases, the patient's progress is slow and unsatisfactory, this strictly lacto-farinaceous regimen must be relaxed in favour of a more mixed diet, and recourse must be had to clear vegetable soups, meat infusions, such as well-made Julienne, mutton, veal, and chicken broths, and raw meat-juice. The foregoing may be improved, as Sir William Jenner long since pointed out, by the addition of the expressed juices of fresh vegetables (carrots, parsley, turnips, celery, etc.), or, on the other hand, by adding a tablespoonful of Valentine's meat-juice to each pint.

Should thirst be a prominent symptom, pure water, albumenwater, barley-water, or home-made lemonade, form suitable beverages.

Hot weak tea, freshly infused with milk, is often grateful to a febrile patient; eggs may be beaten up with this as a medium.

When the vitality is greatly lowered by continuous pyrexia, a little brandy or whiskey may prove of considerable value in promoting appetite and assisting digestion.

The caprices of these patients in regard to food are endless, and will tax to the utmost the resources of the physician in his attempts to devise some variation in the monotony of the diet suitable for them at this stage.

While, in some instances, the course of the pyrexia is continuous, fortunately, in the majority of cases, it is diversified by afebrile intervals. Too much stress cannot be laid on the prime importance of taking advantage of these periods of increased digestive capacity. When they occur, some of the more easily assimilable forms of food should be included in the dietary. Among those permissible in these apyrexial periods are sweetbreads, oysters, lightly boiled fish of the digestible kinds, such as sole and whiting, also pounded and minced meats.

Once convalescence is established, no time should be lost in placing the patient on three good meals a day, with abundance of meat, and, if necessary, a reasonable quantity of alcohol.

In addition milk, if possible, should be taken between the meals, and, if such is distasteful in the daytime, it will often be found acceptable either at bedtime or during the night, and may incidentally promote the advent of sleep in a restless subject.

As regards the form of alcohol most suitable for these cases, I have more faith in good stout than any other variety of stimulant. One pint is sufficient within the twenty-four hours, and it may with advantage be distributed over lunch and dinner. Here I may add that, nauseous and repulsive as it may seem, some patients prefer to take their stout mixed with an equal quantity of milk, and apparently with advantage.

Where stout disagrees, or there are conscientious scruples in regard to alcohol, I have found Horlick's malted milk an excellent substitute, and one, moreover, capable of inducing a notable increase of appetite.

In conclusion, I would emphasize the importance of not restricting these patients for too long a period to an exclusive milk diet.

I am convinced that by so doing their recovery is retarded, a great deal of valuable time being often lost, during which the active progress of the disease might be cut short, and the ultimate degree of crippledom diminished, by a more varied regimen.

Doubtless, the confusion of these acute forms of rheumatoid arthritis with acute rheumatism is largely responsible for the reluctance displayed by many in placing them on a more generous diet. The deplorable results that follow this misconception urgently indicate the desirability of a more timely discrimination between these two joint-affections.

DIET IN THE CHRONIC TYPE.

We pass now to consider the principles of diet in their application to the more slowly progressive forms of the affection. Here, it may be taken as a general rule that good nourishing food should be partaken of in any abundance which stops short of derangement of the digestive functions; or, as Cicero, in his "De Senectute," tersely puts it, "*Tantum cibi et potionis adhibendum ut reficiantur vires non opprimantur.*"

It must not be overlooked that many of these patients suffer from gastro-intestinal derangements which may either have preceded the rheumatoid affection, or have arisen subsequently. In either case it may be taken for granted that no amelioration of the joint condition is to be expected unless these digestive disorders are intelligently and perseveringly treated in accordance with their varied nature.

These patients need carefully selected food, and the importance of thorough mastication and regularity in meal-times should be impressed upon them.

The desirability of postponing the drinking of fluid to the end of the meal, and then only in small amounts, should be emphasized.

The beneficial effect of rest, both before and after meals, for a period varying from half an hour to an hour, for the purpose of promoting digestion and assimilation, should be taken advantage of.

No less important is it that a cheerful atmosphere should be maintained during the actual progress of meals, for it is but too true that " unquiet meals make ill digestions."

The nerve-element in these cases is so pronounced that caution on this point is anything but superfluous, having regard to the inhibitory effects of mental worry and excitement on the secretory mechanisms of the alimentary tract.

With these general considerations we will now enter upon a discussion of the various food-stuffs.

Animal Foods.—Owing to the frequent confusion of the more chronic forms of rheumatoid arthritis with gout, marked restriction of, and in some instances complete abstinence from, meat is enjoined, often with disastrous consequences. The still wide-spread impression that animal food is always undesirable for this class of patients is quite a mistake.

The marked and rapid improvement that frequently follows the introduction of a more generous rule in this respect indicates that meat should be allotted a prominent place in any dietary laid down for a rheumatoid patient.

The general flabbiness and lack of tone in these patients distinctly suggests their urgent need of this variety of aliment, whose energizing and heat-producing qualities are so well ascertained.

Abstention from meat should only be enjoined in the presence of substances in the urine indicative of excessive intestinal putrefaction, meat being, of all articles of food, the chief offender in this respect, because of its marked tendency to undergo putrefaction, with formation of indol.

In the absence of any such indications the following may be taken : Poultry, fresh game, lamb, mutton, beef, fat bacon, and ham. Fish of all sorts—such as sole, plaice, whiting, turbot, brill, cod, fresh haddock—are permissible. To these, if they agree, may be added salmon, red and grey mullet, herring, and mackerel.

If symptoms of dyspepsia make their appearance, the diet must be modified accordingly. The coarser kinds of fish and meat should be eschewed, and the lighter and more digestible varieties taken in preference.

If accompanied by indicanuria and excess of aromatic sulphates, a return must be made temporarily to a strict lacto-farinaceous regimen.

Farinaceous Foods.—Some of the subjects of this disease suffer at times from amylaceous dyspepsia, and are unable to digest large amounts of starchy and saccharine foods, as evidenced by considerable discomfort after meals due to flatulent distention.

In such cases there should be a reduction temporarily in the amount of this class of food—such as bread, potatoes, milk puddings, etc. In their absence, no restriction is called for in this variety of aliment.

Fats, such as fresh butter, bone-marrow, fat bacon, and suet in the form of puddings, are highly desirable; while in this category, if it can be taken, cod-liver oil deserves especial mention.

Vegetables.—The proportion of vegetable food taken will naturally vary with individual digestive capabilities. As a matter of idiosyncrasy certain articles of this class disagree with certain patients, and such peculiarities must be respected. Short of this, no restriction need be placed upon the patient consuming a moderate quantity of fresh vegetables—such as cauliflower, broccoli, cabbages of all kinds, potatoes, vegetable marrow, sea or Scotch kale, green peas, kidney beans, tomatoes, artichokes, spinach, asparagus, Brussels sprouts, lettuce, radishes, celery, onions, mustard and cress. Where possible vegetables should be given in the form of purées, their digestibility being enhanced by this mode of preparation.

Salads should not be partaken of except by those with strong digestive powers.

Fruits.—Apples, pears, plums, when of good quality and cooked, are permissible in moderation; while other varieties may be eaten raw when thoroughly ripe, such as peaches, nectarines, and apricots, grapes, fresh figs and dates; also lemons and oranges, when ripe and carefully selected, may be eaten, and the latter are useful for assuaging thirst in these patients when feverish. In addition, walnuts, almonds, and filberts may be partaken of sparingly.

Beverages.—Tea, coffee, cocoa, and chocolate, when of good quality and properly prepared, are all suitable in moderation; likewise milk, cream, and milk punch.

Of alcoholic beverages, malt liquors are the most desirable. Where these disagree, or are unacceptable, a good red wine—such as Burgundy, or a sound claret—may be substituted. Whiskey or brandy, diluted with Apollinaris or other mineral water, may be taken instead, if preferred; but, whatever the variety of the stimulant chosen, it should only be taken under the direction of the physician, its amount and time of ingestion being strictly fixed.

CLOTHING.

In acute cases, when confined to bed, a long flannel night-dress, buttoning all the way down the front, should be worn. The sleeves should be capacious, and well slit up, with buttons along the outer side, so as to allow of ready exposure of the affected joints, and wide enough to admit of local dressings being easily applied to the inflamed articulations.

In chronic stages the body should be clothed lightly but warmly, woollen garments being worn next to the skin. As Dr. Bannatyne[9] suggests, a thin flannel vest and drawers, with pieces of wash-leather fixed on the inner side of the fabric over the site of the joints, is an admirable plan. It is of great importance that properly-fitting boots of not too heavy a description should be worn. Shoes should be avoided, because of the lack of support to the ankles.

REST AND EXERCISE.

In the acute stages with pyrexia patients are best confined to bed. Too heavy bed-clothing is not desirable. When the knees or ankles are badly affected, it is advisable to insert a cradle beneath the bed-clothes over the lower extremities so as to avoid the pressure and superincumbent weight of the coverings which are so irksome.

It need hardly be said that these patients must be got out of bed as early as possible, and encouraged in every way to use their joints, which otherwise become increasingly stiff.

In the chronic cases, regular exercise, proportionate to their powers, should be insisted upon. The free action of the skin by this means should be encouraged, and when perspiration ensues after exertion it is well for the patient to change the under-clothing and to be briskly rubbed down with a coarse towel.

Great firmness is necessary to ensure exercise being regularly carried out, as it is by no means easy to convince the patient, when in pain, of the necessity of perseverance in this direction if future crippledom is to be avoided. To promote and increase the mobility of the joints, systematic exercises must be done.

Stiffness and rigidity of the fingers must be combated by knitting, piano-playing, and other manual exercises, amongst which one of the most important is the performance of some exercise which involves the movements of pronation and supination of the forearm—such as turning the handles of doors, withdrawing stoppers from bottles, and such like. For the purpose of improving the mobility of the shoulders the use of pulleys and weights is advisable.

Where skilled supervision is possible, mechanical movements according to the Zander method may be undertaken, but never in the acute stages. Among the most troublesome articulations in which to combat increasing stiffness are the knees; and in order to improve the tone of the wasted extensors, patients should be told to sit at the edge of the couch, and attempts perseveringly made to raise the leg so as to bring it and the thigh into a straight line.

Where walking is too fatiguing, the mobility of the joints will be improved by the daily use of a bicycle fixed on an appropriate stand. The patient seated on this can, by making the pedals revolve, do much to improve the stiffness. In many instances, the radius of excursion of the patient's limbs is so limited that it may be necessary at the outset to secure the pedals nearer to the central axis, and for this purpose, therefore, the cranks of the pedals should be perforated at regular intervals, into which apertures the pedals may be screwed, thus securing a greater or less amplitude of the movement. This plan I have found a very useful one; and, as Kolopinski, who speaks very highly of its efficacy, states, it may be necessary at first to strap the patient's feet on to the pedals, or to hold them there by the hand, until sufficient voluntary control over the joints has been regained.

When exercise is impossible, patients must be taken out in Bath chairs, or other conveyances, so that they may gain all the advantages to be derived from fresh air, sunshine, and change of surroundings; otherwise a most unhealthy introspective mental condition results.

MASSAGE AND PASSIVE MOVEMENTS.

In scientifically-applied massage we have no doubt an agent of great value in the treatment of this disease. Its beneficial results are not confined merely to the affected joints and the atrophied muscles; but, as a consequence of the improved circulation, waste products are more readily excreted, the nerve centres gain tone, and a feeling of general well-being is promoted. It is chiefly employed in rheumatoid arthritis to promote the nutrition of the wasted muscles, and to minimize the amount of contracture. In the acute stage, the joints must be left severely alone, but gentle massage of the muscles, including those of the abdomen and chest, can even at this period be employed. In some cases, however, massage produces a condition of great irritability, and insomnia may result. When such is the case, the treatment has either to be abandoned, or its area of application restricted to the limbs, or a portion of them. In very many instances, specially in the earlier stages, the wasting is largely localized to the extensor groups of limb muscles. My own practice is to order the rubber to concentrate the treatment largely upon these areas, subsequently devoting a short time to general massage when such appears to be beneficial.

In the majority of instances it is not desirable that the time devoted to massage should exceed forty minutes; and it should in all cases be followed by an hour's rest. The rubbing should not be practised too soon after a meal. In cases where it promotes sleep it is best undertaken in the late evening.

Passive movements form an important adjunct to massage. As soon as the joints can be manipulated without pain they should be practised perseveringly, but at the same time with gentleness.

Such passive exercises should, as soon as possible, be combined with resistance movements. If the best results are to be achieved from these measures they should be carried on daily, or on alternate days, for long periods; and much of the hoped-for success will depend upon the hearty co-operation of the patient.

DRUGS.

During the acute phases of the disease, of all the various drugs advocated, quinine in one or other of its forms, in my experience, gives the best results. The sulphate is the salt I generally employ, in 2-gr. doses, in combination with dilute hydrobromic acid in order, as far as possible, to prevent the headache that so frequently follows its administration.

Given in this manner it may be taken every four hours, or even oftener, during the acuter stages, and by gradually reducing its frequency to three times a day, it may be continued throughout the more active periods of the disease. Where gastric irritability is present, the salicylate of quinine with the salicylate of bismuth may be substituted with advantage, and may either be given in a cachet, or suspended in a mixture by means of tragacanth, or, better still, by tincture of orange. Should pain and sleeplessness be prominent symptoms, a cachet, containing aspirin gr. v, sulphate of quinine gr. ij, and bromide of sodium gr. xv, should be given three times a day until these special troubles abate, when it should be withdrawn and a return made to the quinine and hydrobromic acid mixture.

In some instances, phenacetin in 5-gr. doses may be substituted for aspirin in the above prescription, when the latter fails to relieve pain, or causes sickness, as it sometimes does. Aspirin and pyramidon taken in equal proportions have also been recommended for their analgesic effects in this disease, but I have not found this combination more effectual than aspirin by itself.

Very rarely recourse must be had to opium or morphia to relieve pain. One advantage of the prolonged administration of quinine is the antipyretic effect exerted by the drug. When, as in rare instances, the temperature remains unusually high or protracted, I have found tepid or cold sponging the most reliable method for dealing with this complication; indeed, so marked has been the relief afforded in allaying the general painfulness and discomfort of such patients, that I have been led to adopt it frequently in cases where the range of temperature has not of itself demanded any special form of antipyretic treatment.

I can therefore heartily endorse Ewart in his belief that light bed-clothing, combined with cold sponging and rubbing, besides being grateful to the patient, are often very effective in controlling the progress and cutting short the duration of these seemingly interminable cases.

Macalister speaks highly of the efficacy of the hydrochloride of quinine in acute forms of the disease. He advises it to be given in 5- or 10-gr. doses with 30 to 40 min. of dilute hydrobromic acid three times a day.

It is of the greatest importance that a daily evacuation of the bowels should be ensured. For this purpose a mild laxative, such as compound liquorice powder, or a pill containing aloin, nux vomica, and belladonna, should be prescribed. Where there are definite signs of intestinal putrefaction, or hepatic sluggishness, the occasional administration of minute doses of calomel, from $\frac{1}{10}$ gr. to $\frac{1}{8}$ gr. every two hours until a grain is reached, are of use. Phosphate of soda as a morning draught also deserves mention in this connection.

Approaching the treatment of the subacute and more chronic forms of the malady, one is confronted with a bewildering choice of drugs, all of which at one time or another have been credited with an almost specific action. Of none of them, as Garrod[10] states, can it be claimed that they possess anything like the certainty of action that attends the use of salicylates in acute rheumatism.

I think I may say I have tried all the various drugs recommended ; and have also, in individual instances, seen all of them, including the much vaunted guaiacol carbonate, fail conspicuously to arrest the progress of the disease. In the acute types guaiacol carbonate does not, in my opinion, possess any advantage over quinine. Having met with better results, as a rule, from the latter, I invariably use it in one or other of its forms in preference. In cases of the slowly progressive type, guaiacol carbonate, especially when combined with iodides, as Luff advises, does good in some instances, but not in all ; and it should, when adopted, be given an extended trial and in gradually increasing doses. As far as I am aware no ill results follow its prolonged usage, while to exhibit it only for short periods is futile.

In cases with more or less marked gastric derangement I have obtained the best results from carbolic acid and its derivatives, combined with alkalies and bismuth. When, by these means and judicious dieting, the stomach troubles were relieved, the joint swellings soon showed a corresponding improvement.

Where the intestinal canal appeared to be the seat of the digestive disturbances, no drug seemed to answer as well as β-naphthol, combined with calcined magnesia and wood charcoal. Irrigation of the rectum with boric acid solutions has also seemed to me to be of use in some of these cases attended by intestinal putrefaction.

Arsenic, like guaiacol carbonate, is a most serviceable drug in the more chronic forms, especially those attended by inactivity of the skin, subjective coldness, and much pain. It should, as Fuller[11] long since pointed out, be given in increasing doses and immediately after meals, and it should be persevered with for considerable periods. It may be given alone in the shape of arsenious acid in a pill or cachet, but I usually combine it with the extract of nux vomica, and if nerve symptoms are prominent, a grain of valerianate of zinc or quinine may be added.

Under the influence of such a combination the joint swelling and pains may undergo a very striking improvement. Syrup of the iodide of iron has long enjoyed a deserved reputation, and may be given either alone or in combination with arsenic. In some instances a combination of the syrup of iodide of iron with cod-liver oil is serviceable, especially where emaciation is pronounced and the appetite bad.

McCrae[12] recently has emphasized the value of arsenic and the iodide of iron, and considers, with most others, that its administration should, with intervals, be continued for months, and even years.

In those examples of the disease attended by marked vasomotor phenomena I have frequently obtained good results by giving thyroid gland in 1½-gr. doses. Such patients often complain greatly of coldness of the extremities. Both the peri-articular swellings and the accompanying asphyxial state of the hands and feet may strikingly improve while taking the thyroid substance. I was led to try its effects owing to the fact that in a patient the subject of exophthalmic goitre, for which she was treated at one of the Metropolitan hospitals, the rheumatoid condition appeared shortly after the thyroid swelling had undergone recession. On the supposition that the thyroid was in some way connected with the formation of antitoxins, it seemed possible that the appearance of the rheumatoid condition in quick sequence to its recession might be due to exhaustion of the defensive capabilities of the gland in this respect. I know of no drug that acts so quickly in ameliorating the condition of this type of rheumatoid patients. Caution should be exercised in prescribing it; and its effects carefully watched lest unpleasant symptoms supervene.

Macalister, independently of myself, was led to use the same agent, and in one remarkable case of a young lady with early rheumatoid arthritis with Raynaud-like symptoms, a complete and permanent cure happily took place. It is interesting to note that the patient's mother had suffered from myxœdema. It is, I think, also suggestive that empirically we should have found iodine in its various forms so useful in this disease, bearing in mind the richness and variety of the iodic constituents of the thyroid secretion. Lancereaux, Revilliod, Midelton, and others have also reported well of the action of thyroid substance in rheumatoid arthritis, while Solis Cohen obtained good results with thymus gland in similar cases.

While such results from organotherapy are encouraging, it need hardly be said that its employment is at present in a tentative stage, and should only be entered upon under constant medical supervision, and in selected cases.

When, in these cases attended by Raynaud-like conditions of the extremities, the administration of thyroid gland is followed by unpleasant symptoms, I have often found a course of bromide of soda, with full doses of iron, arsenic, and valerian, very effectual in alleviating the troubles.

Presumably in such cases an undue reflex excitability of the cord centres prevails, and may account for the benefit derived from the bromide salts.

In this connection actæa racemosa in doses of 15 to 30 min. per diem is also of use, especially when painful muscular cramps, worse at night, are complained of. It has been claimed that its efficacy is most marked in women in whom the onset of the disease is traceable to some uterine or puerperal complication.

Another drug, worthy of trial for the relief of these painful cramps, is hyoscyamus in full doses.

When the special requirements of the case have been met, and nothing remains but debility, occasional courses of nux vomica, with or without hypophosphites, are indicated.

In conclusion, I would lay stress on the fallacy of placing complete reliance on any one drug in the treatment of this disease, which presents such varied characteristics. It is far wiser to treat cases on general principles, noting any indications for special lines of treatment that may be present, than to consider all has been done when a so-called specific drug has been prescribed.

LOCAL APPLICATIONS.

In the acute stages cold compresses applied to the joints, as recommended by Ewart, are often of more value than hot fomentations.

In some instances I have found an opium and menthol liniment most soothing. Another application worthy of trial is one composed of equal parts of guaiacol and olive oil; and the addition of methyl salicylate to the former is attended, in my experience, with more marked relief of the pain, and, as Bannatyne states, also appears to reduce the articular swelling. M. Desplats first advocated the use of guaiacol locally to rheumatoid joints, diluting it with an equal quantity of glycerin, the joints being painted and covered with a dry dressing.

For painting on the painful joints, lin. methyl salicylatis is useful. When, as sometimes happens, the patient objects strongly to the odour of methyl salicylate, a combination of amyl salicylate and menthol made up with lanoline may be substituted, and forms an excellent preparation with which to smear the joints until acute symptoms have subsided. In the chronic stages, when joints are not the seat of effusion, careful massage may do much to diminish peri-articular thickening and restore some degree of mobility. The iodide and soap liniment may be utilized in this manner, while if massage is not available or is contraindicated by persistent tenderness of the joints, stimulating and anodyne applications, such as equal parts of the liniments of aconite, belladonna, and chloroform, should be given a trial.

COUNTER-IRRITATION.

Enveloping the joints in hot sand, as originally recommended by Haygarth, is of value. For relief of obstinate articular pain, or neuralgic pains, nothing can equal the application of blisters. These may either be applied on the proximal side of joints, or over the actual site of pain. The relief obtained is very striking, and the frequency with which patients ask for their repetition is a proof of their efficacy. For the relief of neuralgic pains, a blister along the line of the nerve trunk apparently implicated will almost certainly be attended by marked diminution of the pain ; thus for example, a narrow blister applied behind the internal condyle of the elbow-joint will often relieve pain along the inner side of the wrist-joint. The actual cautery is also useful in this connection. Latham, of Cambridge, ably seconded by Midelton, of Bournemouth, strongly advocates the application of blisters symmetrically placed on either side of the spinal column at the level of the cervical and lumbar enlargements, the surfaces being dressed with savin ointment for considerable periods. Repeated applications and prolonged treatment, according to these observers, are necessary if the best results are to be obtained. Having had the opportunity of seeing one of Dr. Midelton's cases both before and after treatment, I can heartily endorse his plea for a more extended trial of this method. Teissier[13] also advocates systematic counter-irritation along the length of the spinal column, either by repeated painting with iodine, or by the actual cautery. He also suggests for the relief of local pains the use of a chloride of methyl spray.

TREATMENT BY HYPERÆMIA.

It now remains to consider briefly the treatment by induction of hyperæmia, either active or passive. Under the former must be mentioned treatment by hot-air baths, such as by the Tallerman-Sheffield apparatus, or by radiant heat-baths, such as the Dowsing variety, in which electric lamps are used for the purpose of raising the temperature. They can be either used locally for the treatment of a single joint—such as the wrist, elbow, or ankle—or the whole body may be exposed. The essential difference between these and Turkish baths is that the head in the former is excluded from contact with the heated air, the patient being enabled to breathe air at the ordinary temperature, while the entire body, with the exception of the head, is exposed to dry superheated air. When used locally the effect produced is one of powerful local diaphoresis and intense cutaneous congestion. A practical objection to local application of the hot-air treatment is that, if continued for some time, general diaphoresis is also induced.

They are chiefly useful for stiffness, swelling, and pain. Inasmuch as many joints are usually involved in rheumatoid arthritis, I have found it best to employ the general method.

Whether used locally or generally, they should be followed by sprays or douches gradually cooled, in order to promote reaction and to increase the activity and tone of the superficial vessels.

Bier's method consists in the induction of passive hyperæmia by means of a rubber bandage. Before application of the bandage the limb above the affected joint should be enveloped in cotton-wool to minimize the discomfort from direct pressure by the bandage. The bandage should not be less than two inches wide, its turns should be superimposed, and it is not necessary for its whole width when applied to be more than four inches. It should be wound round the limb with a tightness sufficient to greatly retard the venous flow, but not to arrest the arterial supply to the limb. Bier advises, in cases of rheumatoid arthritis, that the bandage should be worn for twenty-two hours out of the twenty-four; but Wakefield[14] considers, after trial of this prolonged method, that better results are achieved by its application for shorter periods, the bandage being taken off when the pain disappears, and reapplied on its return. According to this observer, the most important point is that the bandage must not cause pain or paræsthesia, this being an indication of an undue constriction. The pulse should not be decreased in force or volume. It is essential that œdema should be produced, otherwise no good results will accrue, and for its subsequent reduction massage should be employed. Wakefield, who observed the results of congestion in thirty cases of rheumatoid arthritis, found that the chief objection lay in the tenderness of the skin and soft tissues produced by the continuous pressure.

In only one of his series of cases did he fail to produce any benefit. The most striking results were obtained as regards the symptom of pain, which was usually relieved in a few hours after the application, the analgesic effect continuing for some weeks. It would appear, however, to be of uncertain value in this respect. In some instances, on the reappearance of pain, the method appeared to lose all effect, and when relief was obtained it was of shorter duration than previously. In only one instance did the application maintain its initial efficiency.

Stiffness is, as a rule, not much benefited; neither is swelling of the joint. It would appear from these results that Bier is justified in his warning that, though worthy of a trial, too sanguine expectations should not be entertained when undertaking this method of treatment.

Ewart's treatment by "interrupted circulation" also deserves mention. By this mode a condition of reflex hyperæmia is induced. The technique is as follows:—

A piece of indiarubber tubing is applied tightly in a single or double loop over padding to the thigh or upper arm, the limb having been previously emptied of blood by raising it above the level of the patient's body, and by pressure applied along the course of the veins. The constriction by the tube is only kept up for a short time, from thirty seconds to two minutes, when it is suddenly withdrawn, and flushing of the limb with blood results. This alternate treatment of constriction and relaxation is repeated three or four times at a sitting at intervals of some minutes, and the method may be practised twice daily.

According to this authority, relief of pain and diminution of swelling follow. In addition, passive movements of stiffened joints may be carried out during the period of retarded circulation.

TREATMENT BY NATURAL THERMAL BATHS.

The differentiation of thermal from non-thermal waters is purely arbitrary. Peale,[15] in his introduction to the classification of mineral waters, states that "all springs whose mean temperatures are above the mean annual temperatures of their respective localities (no matter in how small a degree) are thermal springs."

It is obvious, therefore, that from geographical considerations they must display variations in temperature. Thus, as this writer remarks, in Alaska and Siberia, springs in which the temperatures only range between 32° and 42° F. and never freeze, are nevertheless termed warm springs. They are only admissible into the category of thermal springs by reason of the fact that the ground in these regions is constantly frozen to the depth of hundreds of feet.

On the other hand, springs of the same temperature located in equatorial regions would, on the basis of the above definition, be termed cold.

For the purposes of classification, however, it has been found desirable to group all springs with temperatures above 70° F. as thermal. These again have been divided into two classes, designated tepid or warm, and hot. The temperatures of the former range from 70° to 98° F., while those above 98° are placed in the latter group.

On the ground of this division we may take it that the only natural thermal springs in the British Isles that call for consideration are the following:—Bath (100° to 120° F.), Buxton (82° F.), Hotwells, Clifton (73° F.).

It is obvious from the above data that Sir Hermann Weber is amply justified in his statement that Bath possesses the only really hot natural springs in the British Isles. The tepid springs of Buxton, as will be seen, are from 20° to 40° F. lower in temperature than the hot springs of Bath.

While various modes of classifying natural waters have been suggested, such as geologic, geographic, chemical, and therapeutic, the most serviceable one in the present state of our knowledge is that based upon the chemical composition of the respective waters. The main objection to this basis of classification lies in the fact that some springs have so large a percentage of certain ingredients that they may, according to the individual opinion of different observers, be entitled to a place in more than one group.

Another obvious difficulty is that alluded to by Sir Hermann Weber, that they are classed "according to their reputed most potent chemical ingredient, and not according to the substance which chiefly predominates in their chemical constitution."

Thus Bath finds a place among the thermal sodium chloride waters of Europe (Peale). On the other hand, owing to the large amount of calcareous matter it contains, it is, and with more reason, ranked as a thermal calcium water (Peale). Indeed, with the exception of Szkleno in Hungary (105·8° to 127·4° F.), it is, I believe, the hottest thermal calcium water in Europe.

Looking at Bath water, not only from the point of view of its chemical constituents, but also having regard to its high temperature, it is classed as an acrato-thermal water of high temperature. In this respect, it compares very favourably with the Continental springs of this type, such as Aix-les-Bains (86° to 120° F.), Wildbad (91° to 109° F.), Lucca (100° to 120° F.). It will be noted that the average temperature of Bath is higher than that of Aix-les-Bains. The amount of sulphur the latter contains is so small that, as Sir Hermann Weber remarks, it may, like Bath, be classed as a simple thermal water. The chief chemical constituents of the Bath springs consist of the sulphates of the alkalies and alkaline earths, together with the salts of sodium and magnesium. Analysis of the gases evolved shows the presence of carbonic acid, oxygen, and nitrogen, with appreciable quantities also of argon and helium.

The amount of gases present, free and in combination, is of value by reason of the excitation of the cuticle produced by them.

The proportions in which the gases were present, as ascertained at the *Lancet* Laboratory, were as follows :—

Carbonic Acid Gas - -	220
Oxygen - - -	120
Argon and Helium - -	161
Nitrogen - - -	9499
	10,000

Considerable quantities of carbonate of iron are present in the water, and as a result of an analysis of the iron deposit, the Honourable R. J. Strutt states : " I have found that the deposit contains radium in appreciable quantities," and in the course of his report states further : " I think there can be little doubt that the helium of Bath owes its origin to large quantities of radium at a great depth below the earth's surface. A little of this radium is carried up by the rush of hot water, and is found in the deposit."

As to the radio-active properties of the Bath waters and their possible beneficial therapeutic effect, I do not propose to speculate, but will content myself with a quotation from that eminently judicial organ, the *Lancet*. In a recent annotation dealing with the physiological and therapeutic effects of radium salts or their emanations, the following passage occurs :—

"The view that the healing power of certain natural medicinal waters is due not so much to the ordinary chemical constituents as to the radio-activity which apparently most mineral waters to a more or less degree possess, becomes strengthened also in the light of recent work, while we may have here an explanation as to why natural mineral waters cannot be imitated so as to give the same therapeutic results.

"The régime and treatment enjoined at health resorts have a favourable effect on the individual, doubtless, in most cases, because metabolism is improved, and it remains to be seen what the influence on metabolism may be of a course of subjection to radium emanations ; one of the points to be determined will be whether any benefit resulting arises simply out of healthy oxidation being promoted by the radium emanations or out of much more complex actions."

The water exhibits a faintly alkaline reaction and possesses a peculiar, though not objectionable, taste.

INTERNAL USES OF ACRATO-THERMAL WATERS.

The good effect of Bath and similar waters when taken internally may possibly be due in some measure to their chemical constituents, but is more probably attributable to the ingestion of large quantities of water at a high temperature.

A beneficial effect is thus exerted through the washing out of the stomach and intestines and the flushing out of the kidneys, so providing for the excretion of any toxic material in the economy.

Having regard to the frequency with which gastric and intestinal affections occur in association with rheumatoid arthritis, one must not lose sight of the fact, abundantly attested by experience, that courses of these waters do exercise a sedative and remedial effect upon these conditions.

By experiments in the *Lancet* Laboratory, it was found that Bath water possessed more powers of dissolving uric acid than distilled water at the same temperature, namely 100° F.

It cannot, I think, be doubted that by the internal use of these waters the activity of tissue-metabolism, as well as the secretory and excretory processes, are favourably influenced. Naturally such effects are enhanced when combined with judicious dietetic treatment and general hygienic measures.

No condemnation can be too strong for the thoughtless manner in which the laity betake themselves to the spa and take the waters without the approval and sanction of their medical attendant.

Quite recently, I had an urgent summons to see an aged man who, on his own initiative, had undertaken a course of mineral water treatment in this place. At my visit I found him to be suffering from symptoms of uræmia, his urine being highly albuminous.

Had he consulted his own medical man before leaving home, he would not have been allowed to incur the risks he rashly subjected himself to. Such instances of indiscretion might be multiplied.

EXTERNAL USES.

The beneficial results of balneotherapeutic measures are evidenced by the increasing number of rheumatoid patients who avail themselves of this form of treatment.

It need hardly be said that the best results are obtained in early cases. It must, however, be remembered that in the acute stage baths are contraindicated. Indeed, at this period, not only does no benefit accrue, but positive harm may result.

Similarly, even in cases which have reached a chronic stage, bathing must not be undertaken during an exacerbation, as otherwise the condition will most certainly be aggravated. The acute stage being over, the sooner the patient is sent to some well-equipped spa, the more likely are the results attained to be satisfactory.

The special therapeutic value of balneotherapy lies in its well-ascertained capability of ameliorating the results or sequels of the disease.

As to the *modus operandi* of these high temperature baths, it is to a large extent speculative. Immersion in such causes increased circulation of the blood through skin and superficial structures, induces free diaphoresis, and through its influence on the cutaneous nerves exerts a sedative effect upon the central nervous system. As a result of dilatation of the cutaneous blood-vessels, and diminished peripheral resistance, the blood-pressure is lowered in the ventricles, with consequent reduction in strength of the cardiac contractions.

It is important that these baths should not be given at too high a temperature, otherwise headache and other unpleasant symptoms result from excessive reflex cardiac excitation.

Amongst other effects are an augmentation of the respiratory movements, following an initial diminution in depth. The tone of voluntary muscles, though perhaps increased initially, is subsequently lowered. By this means relief of the muscular spasm so common in rheumatoid arthritis is effected.

While prolonged immersion, by reason of the induced perspiration, lowers the amount of urine secreted, baths of short duration, at a temperature of 104° or slightly above, promote increased secretion of urine.

As to the effects on the general metabolism, it has been ascertained that under the influence of heat sufficiently prolonged to elevate the temperature of the blood, increased oxidation and augmented excretion of carbon dioxide ensue. Experimental observations under the same conditions, according to Winternitz, indicate that the formation of alexins and antitoxins is stimulated by their means.

While the above brief summary may suffice for the general effects of hydrothermal applications, it is now necessary to indicate the various baths and their modes of employment.

To describe these in detail would transgress the limits placed on this volume. I only purpose naming the main varieties and their suitability for different cases.

In patients who are very crippled or feeble in constitution, it is well to begin with the Berthollet or natural vapour baths, or reclining or deep chair-baths (*Plate XVI*).

In the natural vapour bath, by a convenient apparatus it is possible to expose either the whole body or the affected extremity only to the steam derived from the thermal waters. The temperature of these ranges from 110° to 117° F.

The reclining bath calls for no special description, and one need only refer to the fact that these are provided with appliances for the administration of the so-called under-current douche, the

PLATE XVI.

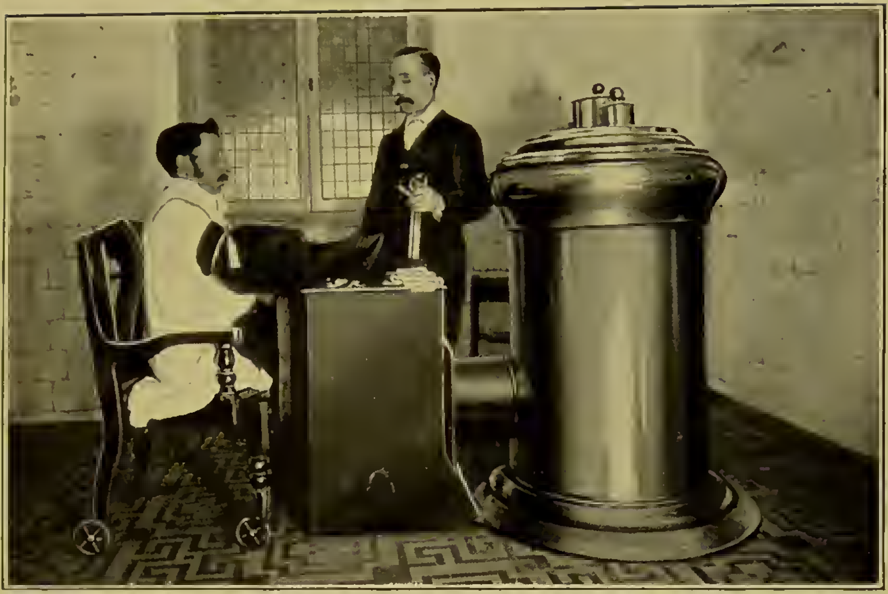


Fig. A.—The "Berthollet" or Natural Vapour Bath, in use for upper and lower limbs.

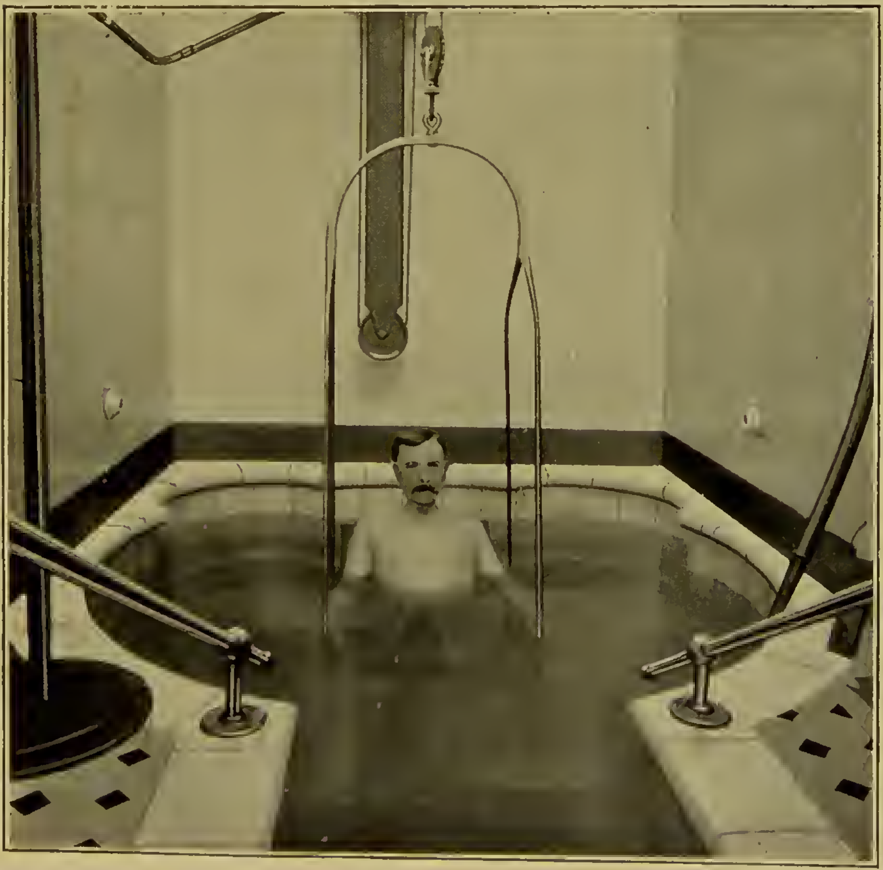
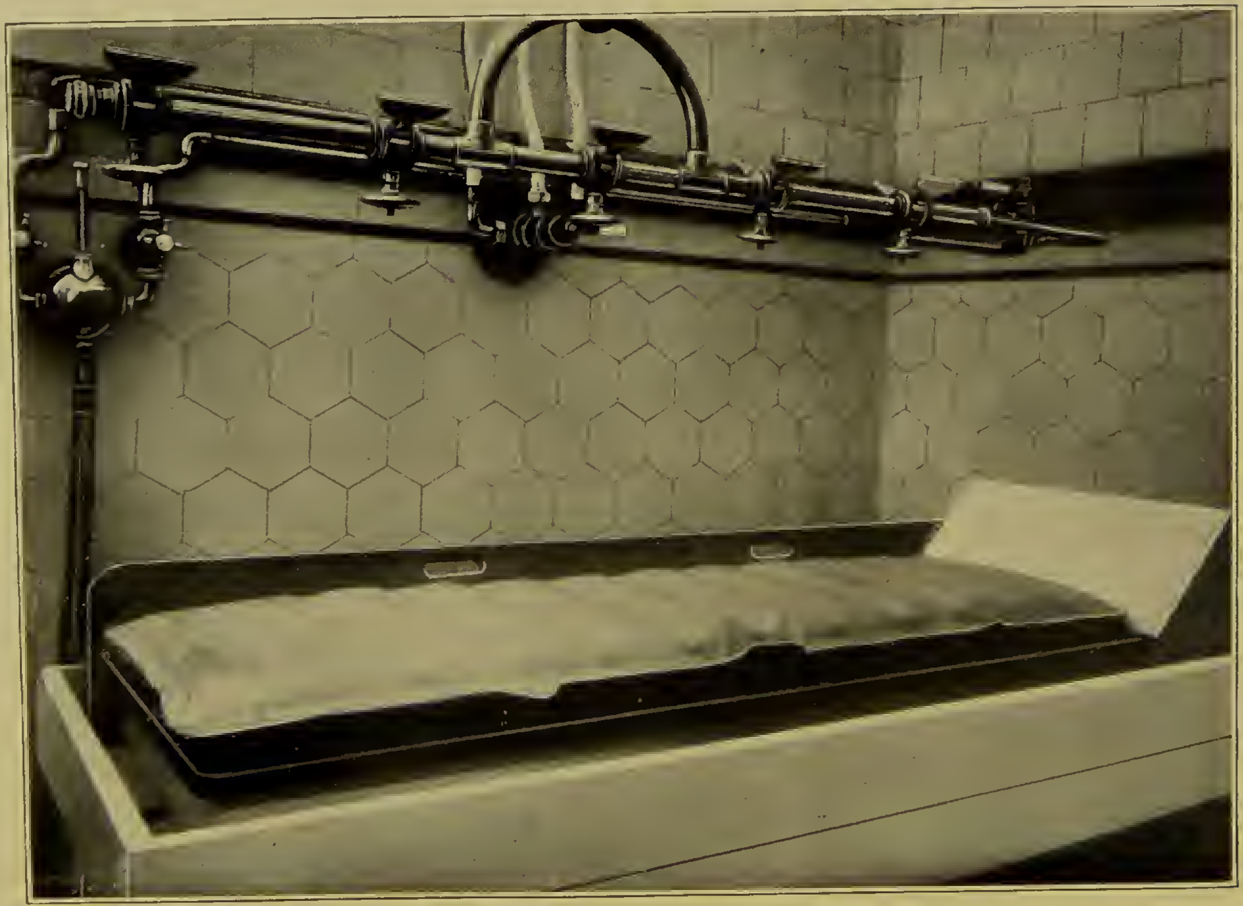


Fig. B.—The Deep Chair-Bath.

PLATE XVII.



Vichy Massage Douche with four descending sprays.

effect of which is so beneficial in alleviating articular and nerve pains when present.

The especial advantage of a deep chair-bath is the provision of an ingenious device for lowering into the water patients who by reason of their infirmity cannot descend the steps into a bath of this description.

For patients of a more robust type, general wet massage, according to the method initiated at Aix-les-Bains, constitutes to my mind the most valuable and efficient method that hydrotherapy possesses for this condition. Under its influence the atrophied muscles gain fresh access of strength, their further wasting is prevented, and the painful muscular spasms are brought under control. In addition, the articular and neuralgic pains are alleviated.

The Bath mineral waters used in this way undoubtedly also exercise a soothing effect upon the nervous system, which is so profoundly affected in this disease.

Incidentally the hot local douching promotes the absorption of peri-articular thickening and checks the tendency to ankylosis from capsular shrinking.

The vasomotor phenomena also undergo amelioration under the improved cutaneous circulation.

In cases sufficiently robust all these baths are with advantage followed by a needle douche, first applied hot and then gradually cooled down. In this way the enervating results of the bath may be counteracted and a tonic bracing effect induced. It is customary here, after most of the various baths, for the patient to be packed. The duration of the packs is from five to fifteen minutes. As a rule 8 to 10 oz. of the mineral water is slowly drunk during the period.

The most modern facilities also exist at Bath for the administration of massage according to the Vichy system (*Plate XVII*), which has one advantage over the Aix system in that the patient is massaged in the recumbent position, thus ensuring that complete muscular relaxation which is so essential for its adequate performance.

Facilities for rectal irrigation with the mineral water have been available in Bath for some years.

It is gratifying also to note that the more satisfactory technique for intestinal irrigation as pursued at Plombières will shortly be installed at Bath. The waters of Plombières, like those of Bath, belong to the acrato-thermal group. From the point of view of their temperature, Bath thermal springs approximate more closely to those of Plombières than those of any other British spa.

The excellent results that have followed treatment by intestinal irrigation in rheumatoid arthritis justify one in expecting that their addition will prove a valuable adjunct to our treatment of the affection.

The duration of a course of spa treatment will vary in individual cases, and no fixed rule is possible. In a moderately severe case a month or six weeks should be sufficient, and when possible should be followed by a visit to some bracing health resort.

Bath is usually classed as a winter resort, and by reason of its climate and situation is admirably adapted as a place of sojourn for these patients from September to June.

In the summer, spas at a higher altitude, such as Buxton, Harrogate, and Llandrindod Wells, possess undoubted claims for consideration, whilst Strathpeffer deservedly enjoys repute.

Woodhall Spa with its iodine springs also deserves mention, while of foreign thermal spas there are a large number available, to mention only a few, namely, Aix-les-Bains, Plombières, Wildbad, Schlangenbad, Lucca, Teplitz, etc.

In conclusion, without unduly lauding the efficacy of hydrothermal treatment in this affection, one may truthfully say that it holds its own as one of the most valuable of the therapeutic agents we possess.

That astute physician, the late Dr. Fuller, when dealing in his classical treatise with the balneotherapeutic treatment of rheumatoid arthritis, devoted a section to dispassionate criticism of the individual merits of the various British and Continental spas. His final words run as follows :—

"But observation has convinced me that the pallid victim to rheumatic gout, who suffers from cold, and is unable to bear the stimulant effect of the sulphur waters, will usually experience more relief from a month's residence at Bath than from a much longer sojourn at any other watering-place."

ELECTRICAL TREATMENT.

For the relief of neuralgic pain, the *continuous current* is of value, the positive pole being placed on the painful spot. Abrupt makes and breaks of current should be avoided, and alterations in the strength of the current must be made very slowly through the medium of an adjustable resistance. The positive electrode should be moved slowly over the painful area.

In joints unduly sensitive to massage, a reduction in tenderness may be procured by the previous passage of a *faradic current* through the joint for ten or fifteen minutes, as Drosdorff has demonstrated. After such, massage of the affected part can be tolerated.

A favourable influence on both local and general metabolism in rheumatoid arthritis has been claimed for *high-frequency currents*. According to Pfahler,[16] the local beneficial effect is attributable not only to the passage of electricity, but also to the counter-irritant effect of the multiple sparks from the glass electrode, also to the violet rays, together with some slight amount of X rays. I have found it a useful accessory in the combating of pain—neuralgic and articular—in these cases. Landis[17] also speaks well of its effects in this disease.

Cataphoresis.—As Pogret[18] many years ago asserted, medicated fluids can by this method be conveyed through the tissues from anode to kathode.

It is, however, to Stephane Leduc that we are indebted for a scientific explanation of the therapeutic value of this method of treatment.

This observer advocates the use of chlorine ions to promote the absorption of fibrous tissues, and salicylic ions for the treatment of rheumatism and neuralgias.

In the opinion of N. S. Finzi[19] chlorine ions are probably of use for sclerolysis if passive movements are performed immediately after the application.

Large pads saturated with a solution of ammonium or sodium chloride are used, a current of 50 to 80 milliampères being passed for one hour three times a week, or, better still, according to this observer, for half an hour every day.

The halogen chiefly employed for medical ionization in rheumatoid arthritis is, however, iodine, the use of which is strongly advocated by Tubby and others. The liniment is painted over the joint, and a constant current of 10 milliampères is passed through the articulation for ten to fifteen minutes.

Diminution of stiffness and increase in mobility of the joints are, it is claimed, attained by this procedure.

Begg,[20] writing on the value of cataphoresis in rheumatoid arthritis, speaks strongly of the relief of pain.

He advises the use of a large negative electrode, 4½ by 2½ in., made of zinc, and laid on chest or back. Button-shaped electrodes of various sizes were used for the positive pole, which was kept dripping with a saturated solution of bicarbonate of potash. Reduction of articular swelling and pain were the results claimed by this observer to have been effected.

N. S. Finzi, who has devoted special attention to medical ionization, states that in common with others he had observed some improvement following the use of salicylic ions in rheumatoid arthritis.

The most encouraging results, however, are, he claims, to be obtained by using lithium and iodine in combination, the former at the positive and the latter at the negative pole.

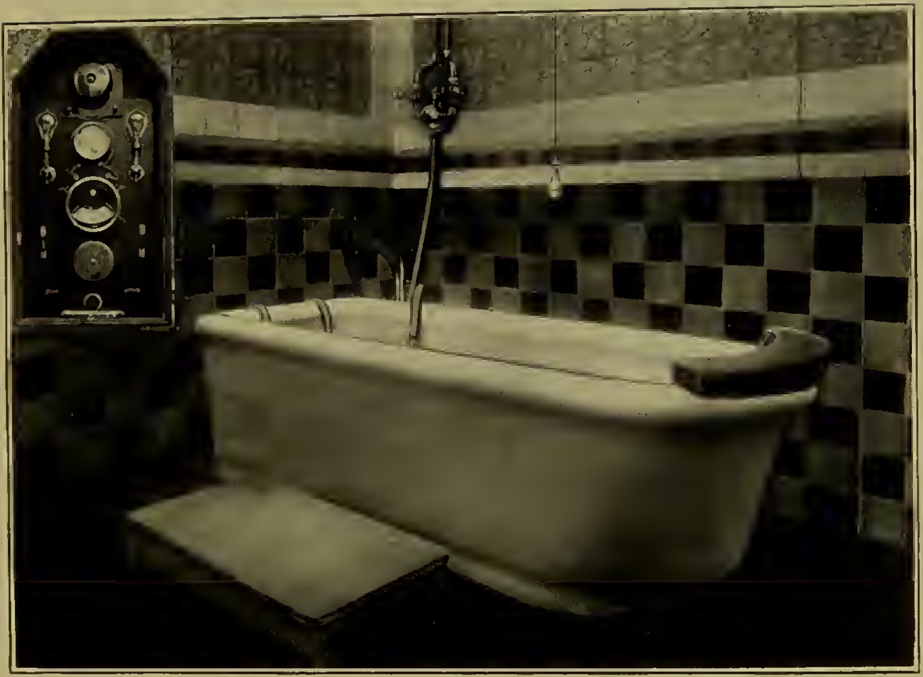
In discussing the possibilities of this method of treatment in rheumatoid arthritis, Finzi, though he does not consider it probable that bony deposits and bony ankylosis can be thus disposed of, yet is hopeful that the cavities so frequently observed in the bones in this disease may possibly be made good by the deposition of new bone under the influence of this treatment.

To obtain the best results from cataphoresis, the most careful attention must be paid to the technique, and those interested cannot do better than consult Finzi's recent contribution on the uses and possibilities of medical ionization.

Hydro-electric Baths.—These are of two types, the *hydro-electric bath* (*Plate XVIII*) and the *four-cell bath of Dr. Schnee* (*Plate XIX*).

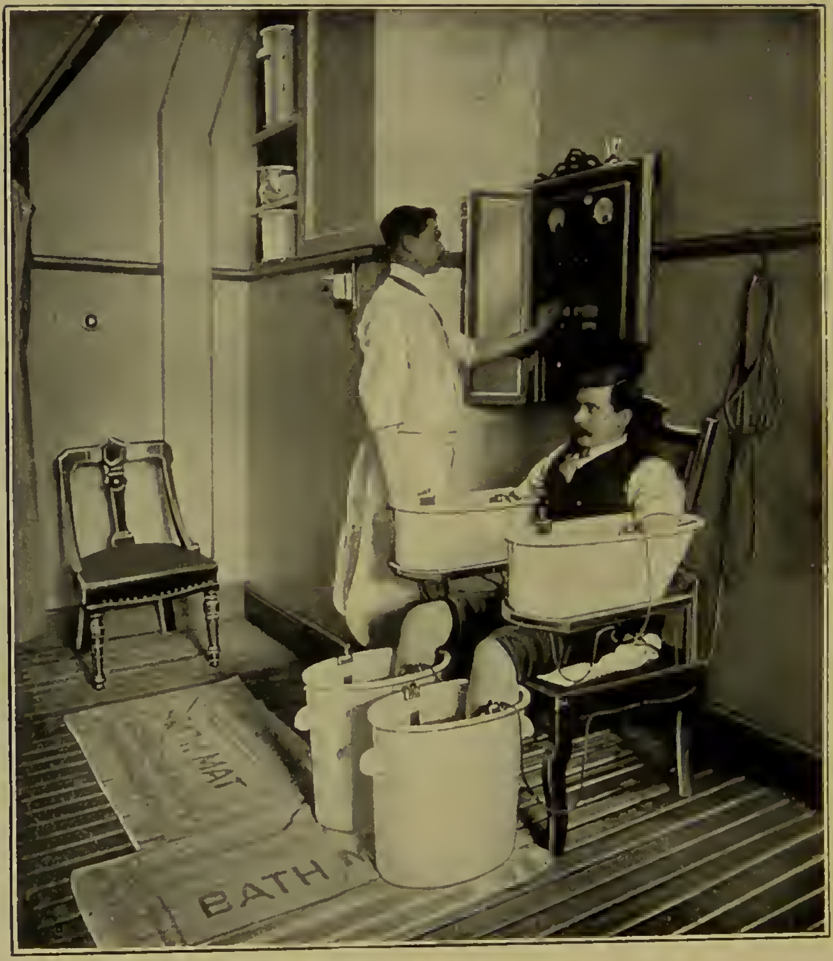
In the former the whole body is immersed. The bath is made of porcelain or wood, and elevated on ebonite blocks.

PLATE XVIII.



The Hydro-electric Bath.

PLATE XIX.



Schnee 4-cell Bath.

The currents utilized are either continuous or alternating, faradic-galvanic or galvano-faradic. In the bath are placed large copper-plate electrodes, the anode being situated at the head, the kathode at the foot. The current thus traverses the body along the line of least resistance from the head to the feet. The current (4 to 5 milliampères) should be switched on before the patient enters the bath, and not until he has risen out of it should it be switched off, so as to avoid unpleasant surprises. Sudden and erratic changes in the strength of the current while the patient is in the bath should also be carefully guarded against.

The temperature of the bath should range from 97° to 100° F., and it should not exceed. twenty minutes in duration. In Bath and other spas the natural thermal water is utilized. The invigorating and tonic effects of this bath are very appreciable in cases of rheumatoid arthritis. To secure the best results, the patients must be the subjects of the disease in an early stage, and it is useless employing this mode of treatment unless the baths are given a prolonged trial. Lewis Jones speaks highly of their value in the treatment of this affection.

Schnee's 4-cell Bath.—In this bath only the hands, forearms, feet, and legs are immersed, each in a separate porcelain cell. The galvanic, faradic, or sinusoidal currents can be employed.

Many advantages are claimed for this bath over the general hydro-electric form, such as the complete utilization of the electric current, whereas in the latter the patient's body is only traversed by one-eighth of the total quantity, the rest being carried away by the water. Again, the erect position carries with it less fear of cardiac depression, which in the full bath sometimes occurs, partly as the result of fright and partly because of the added weight of the water (though the water in a well-ordered bath should never be deep enough to cause this mechanical hampering of the heart's action). It saves trouble because it does not involve complete undressing. This bath is constantly used for the treatment of rheumatoid arthritis, and for the above and other reasons is by some preferred to the general bath. Where one joint gives special trouble, this bath obviously presents conveniences for local or sectional application.

Electric Hot-air Baths.—Under the section on hyperæmia, we have before indicated that the active form of this condition may be effected in various ways, and we propose here to deal briefly with the chief methods now in vogue for the attainment of this purpose. The two varieties of apparatus that call for special mention are the Greville and the Dowsing.

In the *Greville* or *non-luminous* variety (*Plate XX*) the heat is attained by the passage of an electrical current through naked wires. These are made of non-oxidizable metals or a compound of metals. The porcelain insulators encircled by the wires are attached to the back of the generator, the front or inner side being made of perforated sheets of aluminium.

Very high temperatures are capable of being generated by this apparatus, as much as 400 to 500° F. being attainable. The temperatures at which they are generally used are from 200° to 300° F. By a convenient arrangement provision is made for general, sectional, or local application of hot, dry air.

Dowsing or Radiant Heat Baths.—In these, the luminous radiant heat is obtained through the medium of special electric lamps, fixed in highly burnished reflectors of convenient form. By their means it is possible to direct the heat-rays on to either the whole or a selected area of the body.

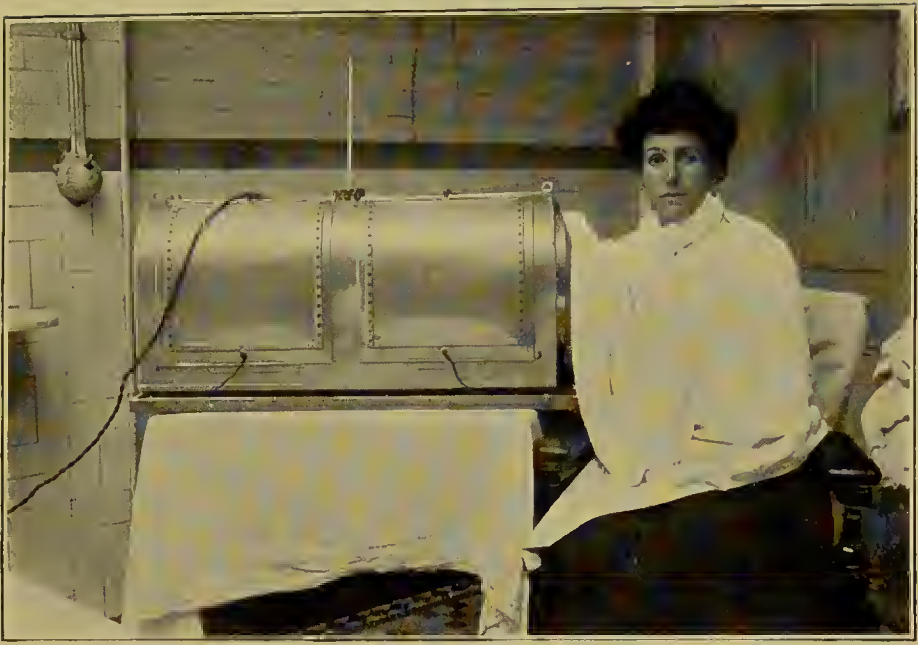
The distinction between this bath and the former is that in the Greville the non-luminous rays beyond the red in the spectrum alone are used, while in the Dowsing the light rays are invoked.

In the Dowsing system temperatures varying from 150° to 232° F. are employed for general exposure, while for local applications considerably higher temperatures may be used with advantage.

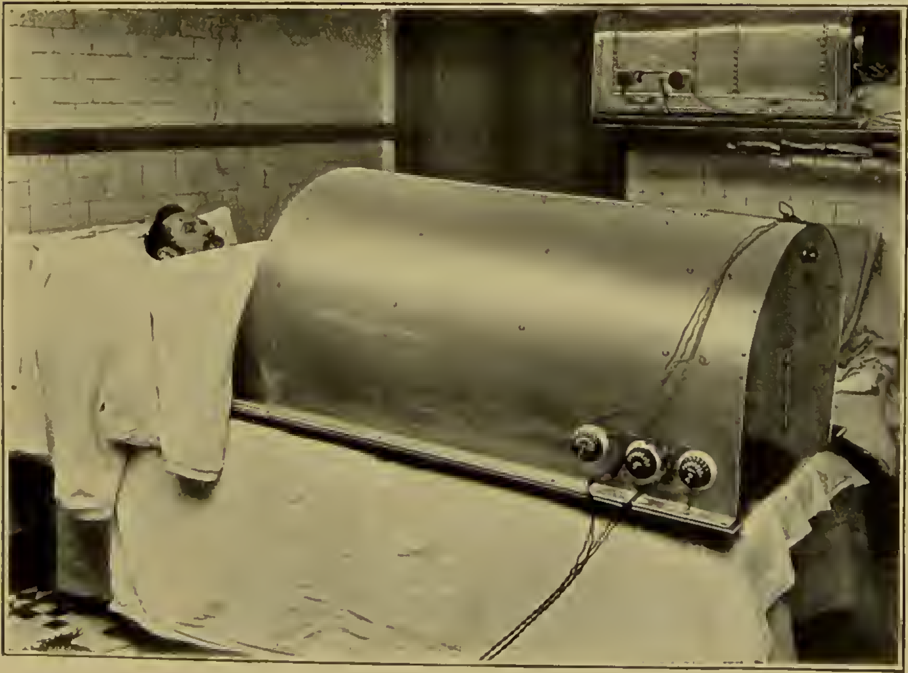
In Bath an alternation of one or other of the above modes of treatment with Aix-massage and other hydrothermal procedures is sometimes adopted. By some, the combination of the two methods is considered to achieve better results than either pursued separately. In the case of the Greville, I invariably order the bath to be followed by a thermal needle douche gradually cooled down.

According to the late Douglas Kerr, even during the course of acute attacks of rheumatoid arthritis a rapid increase in the mobility of the joints, increase in weight, and sense of bodily well-being are produced by a course of Dowsing baths.

PLATE XX.



Greville Apparatus for Arm in use.



Greville Apparatus for Whole Body in use.

Personally, I have never been disposed to submit a patient in the acute stage to this somewhat drastic mode of treatment, so that I am not in a position to confirm or modify this statement as to their therapeutic value in the acute stage.

In the secondary or later stages of the malady, attended by deformity, the same observer claimed good results if, in the absence of pain, the joint adhesions were overcome, either gradually or suddenly, and a Riss bath taken immediately afterwards. The deformity was lessened and the flexibility of the joint increased, presuming the baths to be persevered with. From my own observation, I have found exposure to the Dowsing baths of value in relieving the flexor contracture so obstinate in connection with the knees, a greater degree of voluntary extension being possible after a course of these baths.

PEAT, MUD, AND FANGO BATHS.

The two first-named varieties consist of mineral peat and mud, out of which partially solid baths of varying density are produced.

Saline, ferruginous, and sulphurous varieties of the former are utilized. Mud baths, allied in composition and action to the former, are derived from the various mineral waters or sea-waters. The sulphurous variety is especially used for the treatment of joint diseases, those of Acqui being deservedly renowned.

Fango is the name applied to a peculiar volcanic mineral deposit obtained at Battaglia in Padua.

In all these varieties of baths the action is of a threefold character: mechanical, exerted through compression and friction; thermic, through the medium of the heat; and a possible chemical effect by reason of the constituents they contain.

According to Kisch,[21] as a result of his experiments with iron-peat baths at Marienbad, certain bactericidal properties can be claimed for this variety, these being more intense the greater the amount of acid contained in the peat.

The chemical effects of mud baths are not so appreciable as those produced by peat. All these different modes of treatment, used in conjunction with massage and other hydrotherapeutic and dietetic measures, are reported, especially fango, to have given encouraging results in the treatment of rheumatoid arthritis. They promote absorption of exudates, increase the mobility of the joints, and relieve pain.

In reviewing this brief résumé of the thermal and electrical methods of treatment of the disease, one is conscious of having had, through exigencies of space, to omit many practical details. That strict attention to detail constitutes a most important factor in the attainment of the best results is well known to those accustomed to use these modes of therapy. Regarded individually, none of them can be prescribed as specifics for rheumatoid arthritis. But while accepting their limitations, experience abundantly testifies that, in conjunction with general dietetic and hygienic measures, they contribute not a little to the successful treatment of this obstinate affection.

ORTHOPÆDIC AND SURGICAL.

In the acute stages light splints may be necessary to ensure some degree of rest, and to diminish or prevent subsequent contractures.

The tendency to fixation in the disease is so marked that frequent passive movement is imperative, and for this purpose the splints should be removed night and morning. If necessary, an anæsthetic should be given, to permit of its being performed.

In cases where the joint is so severely affected that some degree of rigidity is certain to ensue, an attempt should be made to secure the fixation of the limb in that position which will prove ultimately most serviceable.

The knees, wrists, and fingers should be kept straight, while in elbows and hips a slight degree of flexion should be aimed at.

Aspiration of Joints.—For the removal of pain consequent on distention of the synovial cavity, aspiration has been repeatedly performed with marked and immediate relief. Removal of intra-articular toxins is also effected by this operation, which, if carried out with antiseptic precautions, is free from danger. Subsequent firm strapping of the articulation seems to diminish the chance of re-accumulation.

Incision of the Joints.—For the purpose of removing hypertrophied synovial fringes arthrotomy has been advocated by

Schüller,[22] and Painter and Erving,[23] who have obtained good results in selected cases. Having regard to the underlying pathological condition, it need hardly be said that such should not be lightly undertaken, and not until other modes of treatment had failed, such as firm bandaging or better strapping of the affected joints.

The articulations in which symptoms of locking or recurring synovial effusions may call for operative interference are the knee or ankle.

In a case which afterwards came under my own observation, the rheumatoid condition began in the left knee. Being attended by excessive villous formation with symptoms of locking, the joint was opened and the enlarged fringes were removed. The ultimate result was not encouraging, for the disease rapidly became generalized, and the patient was reduced to complete crippledom. This case shows the need of careful discrimination in the selection of cases for this operation, which can at best be only palliative.

Tubby also advocates the removal of hypertrophied villi. He has not only practised frequently excision of the pedunculated fringes, but in three instances has gone even further, and removed numerous sessile growths scattered over the synovial membrane. The resulting cicatrization of the membrane seems to do away with the tendency to recurrence of villous overgrowth. Locke and Osgood,[24] who have had the opportunity of examining joints previously subjected to this operation, noted that there was no tendency to the formation of fresh villi in the scar-tissues. Tubby considers that no splinting is necessary after the operation. He relies on soft dressing and the application of pressure. Passive movements are undertaken during the second, and active movements during the third week, so as to avoid the formation of adhesions.

Correction of Malpositions.—We have already dealt with the prevention of deformities in the acute stage. When at a later stage fixation of the joints in a vicious position has occurred, the question of rectification, gradual or forcible, has to be considered. Gradual extension of the knee may be effected by the use of a smooth inclined plane as a leg-rest. It consists of a piece of wood 36 in. long by 9 wide. This is supported at its lower end by another piece of wood of sufficient height to raise it about 4 in. from the ground, and at its upper extremity by a similar piece of wood of sufficient height to raise its edge to the level of the patient's chair. The flexed limb being placed on this inclined plane, the weight of the limb forces the heel downwards, and gradual extension is thus exerted. This should be resorted to for twenty minutes or half an hour three or four times a day. At first painful, the discomfort gradually diminishes, and when combined with hot-air baths or hot douching will often prove an effectual means of straightening the contracted knees in the less inveterate cases. Hoffa, I note, strongly recommends gradual extension by splint-apparatus in contractures of the knee.

Forcible Rectification.—This has been frequently attempted under an anæsthetic. It is usually called for in the case of the knee-joints, but in my experience has been disappointing in its results. In most instances the ensuing pain in the articulation has been so great that the splints or plaster employed to retain the limb in its new position have had to be removed. Even when the pain can be tolerated, the limb subsequently lapses into its former more or less flexed condition.

Hoffa[25] emphasizes strongly the danger of fat embolism after forcible rectification.

Goldthwait[26] is of opinion that when adhesions have formed in the joints these should be broken down and deformities corrected.

Locke and Osgood are accustomed to adopt more radical measures for rectification of malpositions. In regard to the knees, when acutely flexed, they suggest that the first step consists in freeing the patella, otherwise detachment of the patellar tendon from its tubercle may occur. This has actually happened before the adhesions have given way. The fact that too forcible manipulation has caused rupture of the popliteal artery and nerve, with subsequent gangrene, indicates the need for caution. They have found that the contracted posterior capsule of the joint usually relents to manipulations repeated at intervals of one or two weeks.

These observers advocate exposure and division of the hamstring tendons at the time of operation when extension is prevented by their resistance. Also, subsequently to the adoption of a retentive apparatus, if these tendons cause pain from spasm and contracture, they advise their being severed.

After operation, a plaster dressing is applied until the acute symptoms have subsided, when a special calliper splint is substituted.

One of the commonest and most important deformities in rheumatoid arthritis is flat-foot. Inasmuch as the pain due to it may not be limited to the foot but may be referred to the knee and more remote spots, it is often erroneously diagnosed as being of a rheumatic nature. This source of fallacy is enhanced by the fact that considerable swelling of the foot may be present, and thus the mechanical factor is often overlooked.

In such cases the foot-gear should be inspected, and improperly made or ill-fitting boots abandoned. At the same time the sinking arch must be sustained by strapping. If this should prove inefficient, Holland's arched instep socks should be inserted into the boots. These measures may be advantageously combined with massage, alternate hot and cold douching of the feet, and suitable exercises to restore as far as possible the tone of the muscles concerned in the maintenance of the plantar arch.

In dealing with this question of flat-foot in atrophic arthritis, Locke and Osgood lay great stress on the secondary strain thrown upon the knee and hip as a consequence of flat-foot. They recommend forcible rectification under an anæsthetic, and subsequent retention by plaster bandages or splints. The apparatus should be worn persistently for two or three weeks, then replaced in the daytime by an efficient foot-plate or brace, worn in the shoes. They further recommend that the retentive apparatus should be worn at night for a long period.

Where the Achilles tendon is contracted, it should be "fearlessly lengthened or divided," in order to obtain dorsal flexion of the foot to at least a right angle.

Excision of Joints.—This has been performed both for the rectification of faulty position and for the relief of severe and obstinate pain.

In the jaw it may be clearly indicated when ankylosis has resulted; the operation has proved fairly successful.

Should one knee be flexed by contracture, the other remaining healthy, excision of the affected knee may be performed.

Schüller states that in a case of his after excision of one joint, improvement followed in other joints which were affected.

Hoffa, in selected examples, has practised complete extirpation of the diseased joint-capsule, and ablation of the secondarily involved cartilages, with the object of removing the whole area of disease. By this means he was able to correct the malpositions.

Arthrectomy of the knee in one instance was performed by him, with, as he states, only a limited amount of success.

In another case the mobility of the elbow joints was improved by resection of the head of the radius on both sides.

The same author, in a patient with bony ankylosis of the knees, performed cuneiform osteotomy on both sides, with open tenotomies of the flexor tendons. By this the knees, previously acutely flexed, were placed in a position of extension. In the same patient both hip joints were subjected to bilateral subtrochanteric osteotomy. Thus a position of extension and median abduction was secured for both hip joints. As a result, the patient, previously on sticks with perfectly stiff limbs, was enabled once more to walk.

The question of operative treatment in this affection is still in the experimental stage. We do not yet possess sufficient data on the question of mechanical or surgical correction of deformities in rheumatoid arthritis to be able to form a definite opinion as to its utility. Obviously, in view of the underlying pathological condition, cases should be carefully selected, and the operations performed by those with special experience in this department of surgery.

On the other hand, as Tubby rightly remarks, the effects of persistent deformity and impaired motion bring in their trail evils almost as great as those of the disease itself. Consequently the question of operation should not be dismissed as unworthy of consideration.

SPECIAL MODES OF TREATMENT.

Rauenbusch having employed *distension of the joint-cavity by oxygen* as a preliminary to X-ray examination, Hoffa adopted this method. Incidentally he found that in the patients subjected to this mode of examination, improvement of their articular pains ensued, which, moreover, lasted for some days. Encouraged by this result, he systematically carried out insufflation of the joints with oxygen every eight to fourteen days in many patients. In one patient, in which this treatment was carried out over a prolonged period, a very marked improvement in the power of moving the joints followed.

As Hoffa remarks, though it was not to be expected that this treatment could cure the disease itself, yet supposing the condition to be bacterial in origin, it is not inconceivable that it might prove a valuable accessory.

Moreover, the distension of the capsule might tend to check the inevitable shrinkage of the capsule and diminution of functional integrity from agglutination of the articular surfaces.

The Wollenberg-Drager apparatus is utilized for the purpose of insufflation. According to Hoffa's experience, if the technique be properly observed, unpleasant consequences do not occur.

Treatment by Fibrolysin.—As a result of the pronounced destruction and absorption of exudates and cicatricial tissue that followed the intramuscular injection of thiosinamin, this treatment was largely used for the amelioration of cutaneous scars, lupus, etc. While encouraging results were obtained, there were also certain objections to its use. The substance proved unstable, insoluble in water, and injection was followed by pain.

These untoward properties and consequences have been minimised since the introduction of fibrolysin by Felix Mendel, of Essen. This substance, formed by the union of two molecules of thiosinamin with one molecule of salicylate of soda, possesses similar powers of absorbing fibrous tissue.

Consequently Salfeld[27] was led to employ it for the purpose of overcoming arthritic stiffening from fibrous adhesions. He recorded that absorption of exudates, decrease of swelling, creaking, and pain, with increased mobility, resulted.

S. Gara[28] also speaks very highly of its value in restoring movement in previously rigid joints. He narrates the case of a knee joint fixed in a position of full extension, in which, after seventeen injections of fibrolysin, flexion to an angle of 90° was easily possible.

In another instance of complete ankylosis of the elbow joint at an angle of 145°, after the same number of injections the joint, previously rigid for over a year, could be bent. Subsequently other observers have reported favourably of its capabilities in this respect.

Although still in the experimental stage it would appear that we have in this substance a valuable means of combating the immobility resulting from fibrous adhesions, even when of long standing.

It must of course be combined with general and local treatment, such as hot douching, massage, and extension by the gradual method. The preparation manufactured by Merck is put up in an aqueous solution of 15 per cent strength in sterile glass ampullæ each containing 2·3 cc. It should be injected deeply into the gluteal muscles, as when used hypodermically it causes pain, irritation, and swelling, and is less rapidly absorbed.

The injections should be administered once or twice a week, and in view of the fact that unpleasant constitutional disturbance may follow, it is prudent to commence with half the full dose. A sharp rise in temperature with rigors has been noted by several observers to follow an injection.

In a case under the care of Begg, of Bath, severe pain with rigors and vomiting followed two separate injections at a week's interval; and similarly in a patient under Thomson, of Bath, rigors and high temperature followed each administration of the drug.

A recrudescence of pain and swelling in the joints sometimes follows. In conclusion, the treatment is not suitable for either acute or subacute forms of the disease, and should only be undertaken in cases where the joint trouble is quite quiescent, with the object of restoring the mobility of the rigid joints.

Treatment by Vaccines.—The only notable contribution that I have been able to glean from the literature is one by Painter,[29] of Boston.

Briefly considered, the results are as follows: Fayerweather, as was noted in the section on bacteriology, isolated from the knee joint of a patient, operated on by Painter, a micro-organism. From the description in Fayerweather's article, Schüller came to the conclusion that it was identical with his bacillus.

Cultures of this organism having been preserved in the Tufts

Pathological Laboratory, Leary was enabled to prepare a vaccine. An attempt was then made to diagnose and treat the type of chronic polyarthritis with which Schüller claims that his bacillus is constantly associated.

After a year's experience of the application of the opsonic test for this bacillus, Painter noted the following facts: A characteristic serum reaction was obtained in the blood of nearly all the patients presenting the clinical features of this type of polyarthritis. As regards the results of treatment, Painter writes as follows: "I think it is fair to say that there has been no considerable degree of improvement in any case, at least none which is not frequently seen in cases treated by other methods." He considered, however, that the uniformity in the opsonic findings obtained in these cases, clinically of one type, was noteworthy. If to this be added the further fact that a local arthritic reaction succeeded inoculation with the vaccine prepared from this organism, similar to that following tuberculin injections in tubercular joint disease, it would seem, as Painter says, that a stimulus has been given to further investigations along these lines.

In conclusion, as was seen in the section on bacteriology, the evidence in favour of the direct bacterial infection of the joints is not conclusive. Also there are many indications that the toxins are derived from some remote site, possibly the gastro-intestinal tract.

It is here worthy of mention as bearing upon this line of treatment, that in botulism Van Ermengen[30] has found that the symmetrical motor palsies and gastro-intestinal symptoms characteristic of this disease are the effect of a specific toxin formed by an anaerobic bacillus. Subsequent investigations have demonstrated that by means of this toxin it was possible to induce the development of an immunizing toxic serum.

It is interesting therefore to record that Cammidge, in cases of intestinal indigestion with toxæmia, has been able, by inoculations of vaccines prepared from cultures of the predominant micro-organisms in the fæcal excretions, to ameliorate the toxic symptoms. In some instances the same encouraging results were obtained by administration orally on an empty stomach of the same vaccines.

Having regard to the tendency to attribute rheumatoid arthritis to a gastro-intestinal toxæmia, it is to be hoped that further researches will be conducted along these lines, in order to furnish us with a more rational, and hence more effectual, method of treating this deplorable affection.

CONCLUSION.

In bringing to a close this chapter on the treatment of rheumatoid arthritis, it will not I think, be out of place to emphasize the importance of securing the most cheerful surroundings possible for the victims of so depressing a malady. One should be careful to maintain a cheerful attitude in their presence, and thus foster and keep alive in them the hope of eventual amelioration, or haply recovery, being mindful of the old saying, in which there is considerable truth, " A merry heart doeth good like a medicine, but a broken spirit drieth the bones." One does this with less diffidence than formerly, in view of our increasing recognition of the potency of mental factors, not only as predisposing to disease, but also as valuable means of combating its effects. As we have pointed out, there is much to indicate that mental worry, grief, and strain play an ill-understood, though none the less palpable, share in the production of the malady. Equally certain is it that depressing environmental conditions militate strongly against the success of its treatment. To such a patient, stricken, perhaps, in his youth by a greater or less degree of crippledom, it may well be that the vista of the coming years carries despair to a lively imagination. Held in bondage by their distorted joints, one wonders in truth that they do not more often, like Hezekiah of old, turn their face to the wall.

> " Sweet recreation barred, what doth ensue
> But moody and dull melancholy,
> Kinsman to grim and comfortless despair ? "

The most effectual antidote for such morbid introspection is to deflect their attention into some objective channel. By so doing, we may relieve them of something of that painful sense of dependence and uselessness so galling to one of still active mind and energetic disposition.

It will not then, I think, be incompatible with our physicianly dignity to ascertain our patient's mental proclivities, and so assist and encourage them to take up hobbies. These will vary with individual tastes and capabilities, and I would only lay stress upon the eminent suitability for these patients of the mechanical crafts—wood-carving, repoussé work in brass and copper, ornamental bookbinding, and such like. These are beneficial not only in maintaining and increasing the mobility of stiffening joints, but also play a no less important rôle in the mental recreation they afford to the unhappy rheumatoid sufferer, who is but too apt to feel that there is for him no niche in Life's manifold activities.

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SECTION III.

OSTEO-ARTHRITIS.

SECTION III.—OSTEO-ARTHRITIS.

CHAPTER XI.

ETIOLOGY AND PATHOGENY.

MUCH remains to be done before we can claim to be in possession of anything like an exact etiology of osteo-arthritis. Of the more modern writers Goldthwait[1] contents himself with the laconic statement that "The etiology in this type of disease is not known, but cold, exposure, strain, and other injuries are factors of importance."

Teissier[2] also, in his recent work on the subject, seems inclined to assign an equally important rôle to cold and humidity, but it can hardly be doubted that these environmental circumstances are inadequate in themselves to evoke the disease, being probably of the nature of predisposing rather than actual causes.

As regards the mere anatomical lesions of the affection, it cannot be claimed that they present any specific characteristics, and it appears certain that this condition of osteo-arthritis is one that may be arrived at by a variety of pathological routes.

Thus in a few isolated instances the downfall of an articulation through this disease seems to have come via the blood-vessels, through disease of their coats, or alterations in their fluid contents; in others some morbid change in the central nervous system appears primarily responsible; or yet again, we may have to fall back upon some obscure disorder of metabolism, some long-forgotten trauma or obsolete inflammatory process, to account for its genesis.

In discussing the first of these etiological possibilities it will readily be granted that for the maintenance of tissues in a state of integrity, among other factors, it is essential that there be no deficiency, either qualitative or quantitative, in their blood or lymph supply, otherwise the nutrition of the related structures inevitably suffers. In this connection, therefore, it is interesting to observe the stress laid by Billroth[3] and other early writers on the association of osteo-arthritis with arteriosclerosis. As the old term "senile arthritis" connotes, the subjects of the disease are often persons well stricken in years, displaying, amidst other signs of senility, the tokens of widespread arterial degeneration, in which it is quite possible that the nutrient arteries of the joint structures participate.

This implied suggestion of some obscure relationship between the two conditions is not restricted to the older physicians, for it finds expression also in the writings of more modern authorities, Teissier, for example, having drawn attention to the frequency with which chronic joint disorders, closely resembling the various forms of arthritis deformans, develop in the subjects of arteriosclerosis. Lancereaux[4] also has observed similar affinities, and points out this peculiarity, that in such cases the joints of the lower extremities are more commonly affected. In addition Nathan[5] considers that arteriosclerosis is causally related to what he terms the trophic as opposed to the infective variety of osteo-arthritis.

Admitting the relatively common association of these two morbid entities osteo-arthritis and arteriosclerosis, the question now arises as to whether there is any direct evidence that osteo-arthritic changes may supervene secondarily to disease in the related vascular structures, and it must be admitted that such, if not entirely lacking, is at any rate very scanty. The researches of Wickham Legg,[6] it is true, have demonstrated that in hæmophilia osteo-arthritic changes may develop as a result of repeated intra-articular hæmorrhages, but unhappily for our needs, these observations, valuable as they are, do nothing more than establish the possibility of this sequence of pathological events; for what analogy, clinical or otherwise, can be drawn between the youthful victims of hæmophilia and the senile subjects of osteo-arthritis? It is more germane to our line of thought to remember that in aged people the subjects of arteriosclerosis, the nutrition of both bones and cartilage suffers, but still more pertinent is the observation made by Hoffa and Wollenberg[7] while engaged in a recent study of osteo-arthritis. Thus in one of their cases histological examination of the synovial membrane on the cartilaginous border revealed the presence of endarteritis obliterans of typical character, and the suggestion made by these observers that the narrowing of the lumina, by obstructing the arterial flow, might initiate nutritional changes in the cartilage, seems most reasonable.

Again, Painter and Erving,[8] in a case of osteo-arthritis of the knee following trauma, and complicated by marked villous hypertrophy, resorted to surgical treatment, during the course of which they removed some enlarged fringes, likewise a bony spur projecting upwards from the margin of the tibial condyle, and noted in a microscopical examination of the removed fringes that the related vessels showed much thickening of their walls, their lumina being in many cases obliterated.

Isolated though these observations be, they are certainly very striking, and carry with them the obvious inference that detailed examination of the blood-vessels of the synovial membrane, bone, and bone-marrow, in cases of osteo-arthritis, might prove a very profitable line of research. Until these or similar exact pathological findings are forthcoming in adequate number, our views as to the etiology of, at any rate, these senile forms of osteo-arthritis must rest upon the broad assumption that the joint structures enjoy no immunity from the impaired nutrition and general lowered vitality incidental to advanced periods of life; in other words, that the condition arises out of some disorder in the nutrition of the bone and cartilaginous structures. Indeed, excluding the wear and tear entailed during a long and perhaps laborious existence, these degenerative processes furnish us with the most likely, if not the only, explanation of those instances in which, though unsuspected during life, osteo-arthritic changes of a mild degree have been found in the joints of aged persons.

The relationship of osteo-arthritis to diseases of the central nervous system next claims attention, for despite their clinical aloofness, lesions anatomically very similar, though magnified to a colossal degree, are to be met with in the arthropathies of nervous origin.

Virchow[9] indeed insisted upon the identity of Charcot's disease with osteo-arthritis, and whether this dictum prove correct or not, the fact remains that up to the present time pathologists have been unable to fix upon any criterion by which the two conditions can be infallibly discriminated, at all events in their earlier stages.

Thus Hoffa and Wollenberg, in their recent treatise on the subject, maintain with Virchow that tabetic arthropathies are none other than examples of genuine osteo-arthritis occurring in the subjects of locomotor ataxy.

Turney,[10] however, while fully alive to the difficulties of the problem, maintains that the two affections are essentially different, even anatomically, basing his differentiation on the fact that in osteo-arthritis the morbid process begins in the cartilage, while in the arthropathies it has its starting-point in the bone, and if this be granted he considers "that the rough similarity between the final results in the two cases becomes easily explicable if we suppose that the vicious circle is, *mutatis mutandis*, the same in both."

Leaving on one side this still vexed question as to the identity or non-identity of the anatomical changes produced in the joint structures, and directing our attention to the same phenomena from the point of view of their relationship to lesions of the nervous system, we soon become cognisant of their fundamental distinctness.

Indifferently, whether dealing with arthropathies in relation to tabes, syringomyelia, or general paralysis, we meet with the same wealth of pathological evidence indicative of widespread nervous disease, and this, moreover, forthcoming with a constancy that precludes any doubt of a causal relationship. As opposed to this glut of evidence in favour of the neurotrophic origin of Charcot's joints, we are confronted in osteo-arthritis with an almost absolute lack of any such pathological data to support the assumption that they are of similar nature. This deficiency notwithstanding, the close resemblance obtaining between the anatomical lesions in both conditions has led a French authority to formulate the theory that all the features of osteo-arthritis could be accounted for by a toxic neuritis of the articular nerve filaments.

Thus Poncet and Leriche,[11] as we know, in a recent study of the pathological anatomy of osteo-arthritis (l'arthrite sèche sénile) have suggested that some cases of this type are of tubercular origin, and the material, both microscopic and macroscopic on which their view was based, having been subjected to the expert scrutiny of Dr. Le Dor, that pathologist advanced as an alternative hypothesis the above modification of the tubercular toxigenic theory. Now the particular site of the nerve lesions responsible for the development of Charcot's joints is still a matter of conjecture, for while some authorities unite in placing them within the cord substance, others follow Pitrès and Carrière in ascribing them to changes in the nutrient nerves of the joints. The balance of evidence, however, is in favour of the former view being the correct one, though there is no *à priori* reason why, given a neuritis of the particular strands linking the hypothetical joint centres with the articular structures, changes identical with the nerve arthropathies should not occur. In opposition to this possibility that a toxic neuritis might be responsible for the genesis of some cases of osteo-arthritis, we have to contend against this fact, that, notwithstanding the great frequency with which we encounter toxic neuritis of all sorts, arthropathies even of the most benign type have rarely, if ever, been seen in this connection.

In conclusion, therefore, while admitting the superficial resemblance that obtains between the anatomical appearances of osteo-arthritis and those of the nervous arthropathies, it is obvious that from a pathological standpoint there is hitherto no evidence that the former is of similar neurotrophic origin, and assuredly there is nothing in the clinical course of osteo-arthritis which could justify the assumption. So remote indeed is its general clinical aspect from that presented by those diseases of the central nervous system in which arthropathies occur, that detailed differentiation is superfluous, and for these reasons, in our opinion, the osteo-arthritides of neurogenic origin are to be regarded as a class quite distinct from that form to which our study is devoted.

Apart from the foregoing relationships, there would appear to be some obscure bond of affinity between osteo-arthritis and disorders of the ductless glands. Modern research, as is well known, tends more and more to show that the internal secretions, so-called, play a most essential rôle in the economy, any alteration in them, whether qualitative or quantitative, creating more or less profound disturbances in the normal current of metabolism. That the skeletal structures, moreover, participate in these untoward consequences is obvious from the misshapen and stunted limbs of the sporadic cretin, carrying with them, as they do, the inference that for the orderly development and growth of the long bones the physiological products of one, at any rate, of these glands—the thyroid—is essential. Again in that peculiar variety of dwarfism, achondroplasia, described by Shattock as a paracretinous condition, catarrhal processes have, in rare instances, been observed in the thyroid gland. Lovett and Nicholls[12] also have noted the association of osteogenesis imperfecta with an unusually small size of the adrenals, while by some, mollities ossium has been ascribed to absence or defect in the internal secretion of the ovaries.

In yet another disease characterized by marked anomalies in the osseous system—acromegaly—we have further and more constant evidences of some subtle relationship or interdependence between disorders of the ductless glands and abnormal disturbances in nutrition of the skeletal and other structures, the almost invariable changes in the pituitary body being not infrequently accompanied by hypertrophy of the thyroid, and more rarely by hypertrophy of the thymus gland. While fully realizing that the exact significance of these correlated changes still remains to be unravelled, it cannot be denied that in some. at any rate, of the diseases above alluded to, alterations in the ductless glands occur with a frequency which distinctly suggests a causal connection between these two groups of morbid phenomena.

In view of our suggestion that some ill-understood relationship exists between osteo-arthritis and disorders of the ductless glands, it is interesting to note on the authority of Oppenheim that changes similar to those of osteo-arthritis are to be met with in the joints of sufferers from acromegaly.

This overlapping of the specific lesions of osteo-arthritis and acromegaly in the same individual is of great interest in view of the constancy with which changes in the pituitary and thyroid gland occur in association with the latter affection. One is led, therefore, to question whether the overgrowth of bone and cartilage characteristic of osteo-arthritis may not in some instances be attributable to disturbances of nutrition in the articular ends consequent upon the withdrawal or perversion of one or more of the internal secretions.

The frequency with which, in women, osteo-arthritis arises at or near the menopause is remarkable, and it is possible that its onset at this period is determined by some modification in the internal secretions of the ovary and thyroid gland.

As we saw when dealing with the opposed condition, rheumatoid or atrophic arthritis, there are indications of some obscure kinship between that affection and exophthalmic goitre, and it now remains for us to note what clinical evidence there is pointing to a similar affinity on the part of osteo-arthritis.

Blake[13], in his work on "Myxœdema and the Goitres," records an instance of the co-existence of Graves's disease and osteo-arthritis in the same patient, and in another osteo-arthritis, goitre, and a deeply bronzed skin.

The same author narrates also the case of a woman, primarily the subject of Graves's disease, who subsequently developed myxœdema and osteo-arthritis of both knees and ankles.

This association of Graves's disease with osteo-arthritis is, in my experience, rare: I have only met with one instance of the overlapping of these two affections; the subject was a middle-aged woman, a sufferer from uterine prolapse and leucorrhœa for many years, and in addition, oral sepsis; during the menopause she developed osteo-arthritis of the knees and Heberden's nodosities, and simultaneously her thyroid became greatly enlarged, giving rise to feelings of suffocation. Trembling of the limbs, and occasionally of the whole trunk, with extreme cardiac irritability, supervened, also slight protrusion of the eyeballs, and pronounced mental excitability and apprehensiveness. I have also seen simple enlargement of the thyroid with osteo-arthritis of the knees.

On the other hand, as touching Blake's example of the association of the opposite condition, myxœdema, with osteo-arthritis, I would just mention that I have frequently observed in women at the menopause, the subjects of osteo-arthritis of the knees, a peculiar change in the consistency of the skin and subcutaneous tissues, more particularly of the lower limbs. The overlying skin is usually harsh, dry, and not infrequently scaly, has lost its suppleness, and is with difficulty lifted up from the subjacent tissues, which to palpation feel tense, elastic, and, like the skin itself, much thickened. No pitting is to be observed on pressure, and the physical sign appears independently of renal and cardiac complications. I have known one instance in which this condition supervened suddenly in the left lower extremity of a woman, who had received an acute fright, during which she experienced a sudden loss of power in the left leg, causing her to fall down; the swelling of the affected limb almost immediately appeared, and was still present when she came under my care some weeks afterwards for osteo-arthritis of the hip and knee of the same side. In this particular instance the limb was considerably increased in size, but in the vast majority of cases it is not so much an increase in size, but an alteration in the consistency of the skin and underlying tissues. It is in the lower extremities that I have chiefly met with this peculiarity, which may involve the whole of both limbs, and intelligent patients have often commented upon the altered texture of the skin in this neighbourhood. The administration of thyroid substance in some instances was quickly followed by a diminution in swelling when present, and also in restoring a more natural consistency to the affected tissues.

Whether this infiltration of the skin and subcutaneous tissues is identical with the diffuse brawny infiltration noted occasionally by Garrod in the fingers of patients affected with multiple osteo-arthritis of the hands, I am unable to say. As to its nature one can only speculate; but objectively at all events it appears indistinguishable from the solid swelling associated with myxœdema. Its localized distribution forbids our identifying it with this affection, but at the same time we must remember that physicians following Hertoghe are learning to postulate the existence of states intermediate between avowed myxœdema, and the normal, "formes frustées," so to speak, analogous to the larval forms of Graves's disease, and presumably due to variable degrees of thyroid inadequacy.

That this, in my experience one of the most common forms of osteo-arthritis, should so frequently arise in women at this critical epoch, when the potent influence of the thyroid and ovarian secretions is being profoundly modified or withdrawn from the economy, gives room for reflection.

Taking these facts in association with the extremely insidious onset of the joint changes, their tardy progress and relatively benign character—features more suggestive of a nutritional alteration in the articular structures than a frankly inflammatory process—one is led to question whether some deficiency or perversion in these particular hormones is not in some subtle way etiologically related to this particular variety of osteo-arthritis.

We turn now to a consideration of the possible infective origin of some cases of osteo-arthritis, for, as was only to be expected from the present trend of medical thought, this view as to its nature has been advanced. That such arthritic changes could ensue in sequence to chronic inflammatory processes of divers kinds has long been known to pathologists.

Kaufmann,[14] writing in 1901, stated that the condition of osteo-arthritis might supervene subsequently to an infectious arthritis, or to a chronic purulent catarrh of the synovial membrane, and this even when such morbid changes were not attended by destructive inflammation of the joint. Indeed, according to this authority the condition might result from a chronic hydrops articuli. More recently Kohler[15] expressed the opinion that tubercular and osteomyelitic affections of the articular ends of the extremities play an important rôle in paving the way for osteo-arthritic changes to develop in the neighbouring joints. Hoffa again cites two instances in which he has been able to trace localized osteo-arthritis of the hip and knee respectively to previous, but long since obsolescent, tubercular disease of the same joints, and the same author, discussing the origin of so-called juvenile osteo-arthritis, considers that the traumatic etiology usually ascribed to it only partially explains its origin, and is more inclined, like Kohler, to attribute it to an overlooked tubercular or osteomyelitic process. In support of this view as to etiology, he refers to two plates in Schreiber's *Atlas of Joint Diseases* as illustrating the occurrence of osteo-arthritis in joints the seat of ancient and long since healed tubercular processes.

The same authority draws attention to the fact that Borchardt records three cases of juvenile osteo-arthritis which were submitted to operation, when a condition of osteomyelitis of the neck of the femur was found in all, while in one the disease was of bilateral distribution. To these may be added the well-known views of many of the French School, notably Poncet and Leriche, who not only believe that the more acute forms of osteo-arthritis are often due to tubercular disease, but would ascribe the same origin to the more chronic forms, such as "l'arthrite sèche sénile." In regard to the proposition that osteo-arthritis may ensue secondarily to infectious forms of arthritis, it cannot be denied that in the more destructive types of the latter new bony formations are to be met with.

These have been observed as the result of pneumococcal, typhoidal, and gonococcal infections, but as Goldthwait has pointed out, if new bone is thrown out in these cases, its formation conforms to that typical of septic osteitis or periostitis, or, as he puts it, the thickening of bone takes place at the point of infection wherever that may be, but does not develop at the margin of the cartilage as in osteo-arthritis. Apart from these differences in the anatomical lesions produced, clinically these destructive forms are very distinct from osteo-arthritis, their much more acute and fulminant course sufficiently differentiating them, and confusion is only possible when they come before one at the expiration of the acute stage, the bony outgrowths then supplying a possible source of fallacy, which, however, careful attention to their previous history will always obviate. Hitherto no organism has been found in osteo-arthritis, although McCrae would assign to this, as well as to all other forms of arthritis deformans, an infective origin; but without denying the possibility of this being so, it would appear to us, from the clinical course of the disease, unlikely that micro-organisms will be found actually within the joint, and that if of infective nature, the arthritic changes are the result of circulating toxins absorbed from some remote focus of infection. It is possible—indeed probable—that certain toxins have a selective affinity for synovial tissues, and may in this way give rise to changes, qualitative and quantitative, in the synovial secretion, with the result that the cartilage, deriving pabulum from this contaminated medium, may suffer in its nutrition.

It now only remains for us to consider the relationship of injuries to osteo-arthritis. Trauma, as we know, has always been assigned a prominent position in the etiology of osteo-arthritis, more particularly the localized forms. It is also noteworthy that while these peculiar arthritic changes may develop in, for example, a hip joint as the result of a single violent injury to the great trochanter or the shaft of the femur, it is equally certain that they may ensue as the result of oft-repeated slight injuries or strains, many of them incidental to special forms of occupation. So trivial indeed is the trauma oftentimes that we are compelled to infer a condition of grave trophic instability in the articulation which finds destruction on such slight occasion. How to explain the exact *modus operandi* of traumata is uncertain, but this we do know, that subsequently to fractures of the epiphyses or even of the diaphyses, attended with laceration of the nutrient artery of the bone, the nutrition of the articular extremities is, at all events for a time, interfered with, and proceeds irregularly. Now, recognizing the fact that in old people the subjects of arteriosclerosis, or it may be of cachectic conditions, the nutrition of the bones and cartilages suffers, it seems at any rate possible that, considering the pathological trend of such individuals, similar nutritional disturbances may follow slight traumatisms. Hoffa lays stress on the frequency with which this condition follows contusion, both violent and slight, and fractures especially those involving the joint, not to mention dislocations, more particularly those unreduced and of long standing. Godlee[16] also has reported cases which suggested to his mind the additional possibility that an injury received even in youth to any particular joint may determine the onset later in life of osteo-arthritis in the same articulation, and this may occur very many years after the injury.

While admitting the frequency with which trauma figures in the history of osteo-arthritic patients, it seems to us that its etiological significance is more in the direction of a determining than an actual cause, there being probably other ill-understood but essential contributory factors necessary for the production of the disease.

A review of the foregoing résumé soon convinces one that much remains to be done before we attain to anything like an exact etiology of osteo-arthritis. At present it would appear that the disease stands, as it were, midway between the infective arthritides on the one hand, and the nervous arthropathies on the other, without displaying any positive characteristics that would justify its inclusion in either group. From the bacteriological side no positive data are forthcoming, and as we have seen also, no changes in the central nervous system have hitherto been detected. Researches along these lines having proved sterile, many authorities have been driven to conclude that it is of dystrophic nature: in other words, a nutritional disorder of the cartilaginous and other articular structures, and one moreover capable of being initiated in divers ways. Some, as we have seen, hold that arteriosclerosis is causally related to some forms of osteo-arthritis, and it seems to us not unlikely that this may be the explanation of some of the senile types of the disease; the resulting failure in the nutrition of the bones and cartilages admitting of the initiation of the joint changes by traumata, violent or trivial. On the other hand, the infective or toxic hypothesis seems more applicable to the osteo-arthritis of young people, whether generalized or localized, but it lacks definite confirmation. I have, I may say, several times, in younger subjects of osteo-arthritis, noted coincident tubercular processes not necessarily pulmonary in type, and I am inclined to think that there is some measure of truth in the contention of the French authors that some of these cases may be the outcome of an "attenuated" tubercular infection, the changes in the joints being due to toxins from some remote tubercular focus, and quite distinct in their characters from the ordinary "tumeur blanche." In those very rare cases of monarticular osteo-arthritis occurring in young children, the possibility of the disease having been engrafted on to an obsolescent tubercular affection of the same joint should always be borne in mind.

In conclusion, one lesson I think we might learn from our review of the scattered etiological factors of osteo-arthritis is, not to confine our study of its morbid anatomy so strictly to the joints, but rather to approach the subject with a more open mind, not only taking into our survey the articular changes, but sedulously searching for anomalies in other directions.

Thus, for instance, the condition of the vessels responsible for the nutrition of the articular structures should be investigated, likewise that of the nerve-strands supplying the joint.

Should the examination of the vascular and nervous elements disclose nothing abnormal, it would seem advisable to wander still further afield in our pathological quest, and perchance discover, as in acromegaly, that the source of the disease, remote from the joints, lies hidden in some as yet unrevealed lesion of one or more of the ductless glands, whose labyrinthine actions and interactions are so essential to the nutritional stability of the organism in all its integral parts.

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Chapter XII.

MORBID ANATOMY.

In this particular variety of arthritis deformans, though all the constituent elements of the articulation participate in the morbid process, the essential anatomical lesions are those produced in the cartilaginous and bony structures, which undergo enlargement or hypertrophy. This overgrowth of bone and cartilage, so constant and characteristic a feature of osteo-arthritis, varies widely, however, in regard to the degree of its development in individual instances, the morbid process finding what may be termed its rudimentary expression in the so-called Heberden's nodosities and reaching its acme in some of the monarticular examples of the disease, such as malum coxæ senilis.

The primary changes, as Virchow long since maintained, take place in the bony and cartilaginous structures, the synovial tissues being only secondarily involved. Subsequently, the periosteum and the ligaments are implicated in the morbid process.

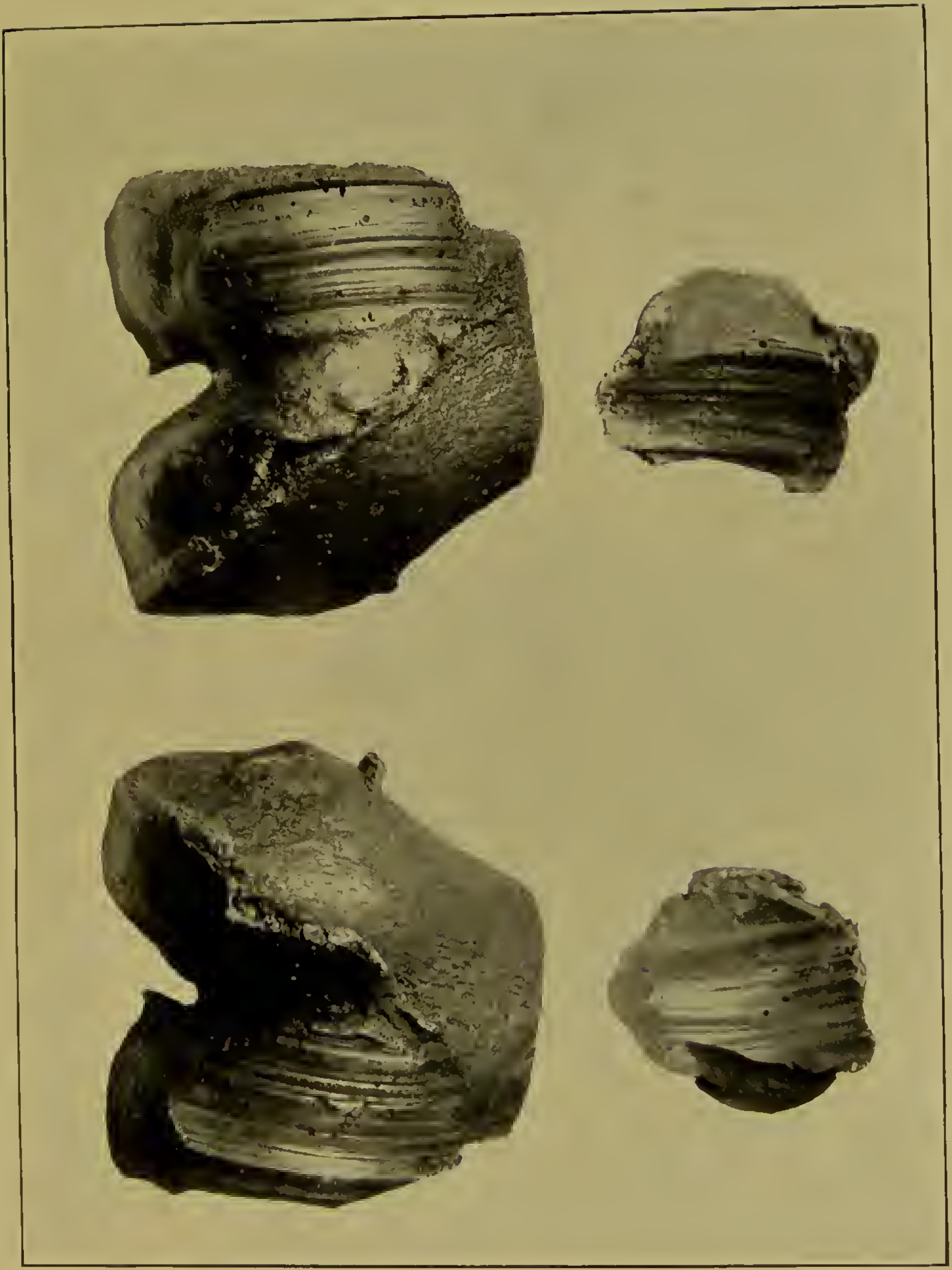
Most authorities, including von Volkmann, Ziegler, Weichelsbaum, and others, confirm Virchow as regards this sequence of the pathological events.

Going more into detail, the intra-articular cartilage loses its normal tint and polish, and becomes softer in consistency.

Histologically its ground substance is seen to be the seat of a process of segmentation, the cartilage cells meanwhile undergoing proliferation, and these latter, released by the fibrillation of the matrix, escape into the joint cavity, where, according to Rindfleisch,[1] they may be found mingled with epithelial débris and occasionally undergoing colloid change.

With the progress of the disease the cartilages, by attrition of their opposing surfaces, become gradually thinned, and the central portion, where the pressure is greatest, being at length completely worn away, the denuded ends of the bones appear. On the other hand, at the margins of the articular surfaces,

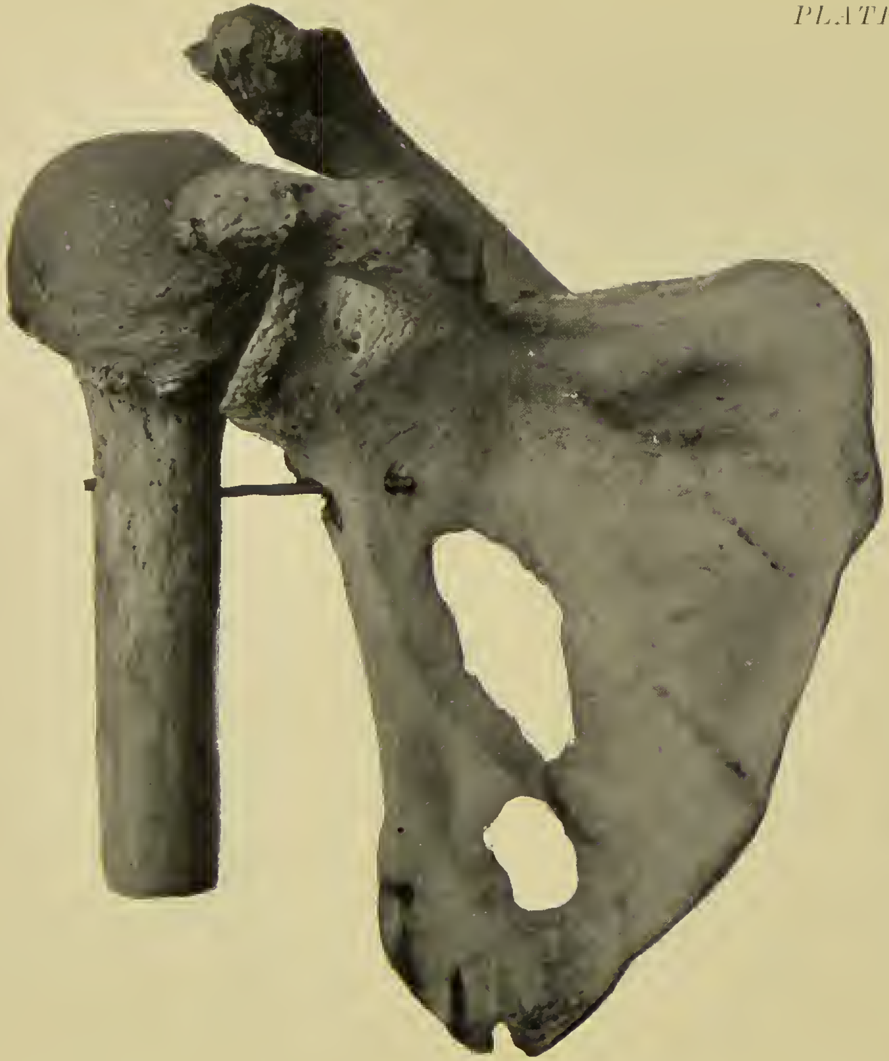
PLATE XXI.



The Lower Extremities of both Femurs together with the Patellæ. Showing the results of long-standing osteo-arthritis. The femurs are symmetrically affected; the patellar surface is smoothly polished and traversed by antero-posterior grooves and ridges. The margin of the articular surfaces is marked by a ridge of new bone. The borders of both patellæ are the seat of new bony formation, and their articular surfaces are polished and grooved in exactly similar manner to those of both femurs.

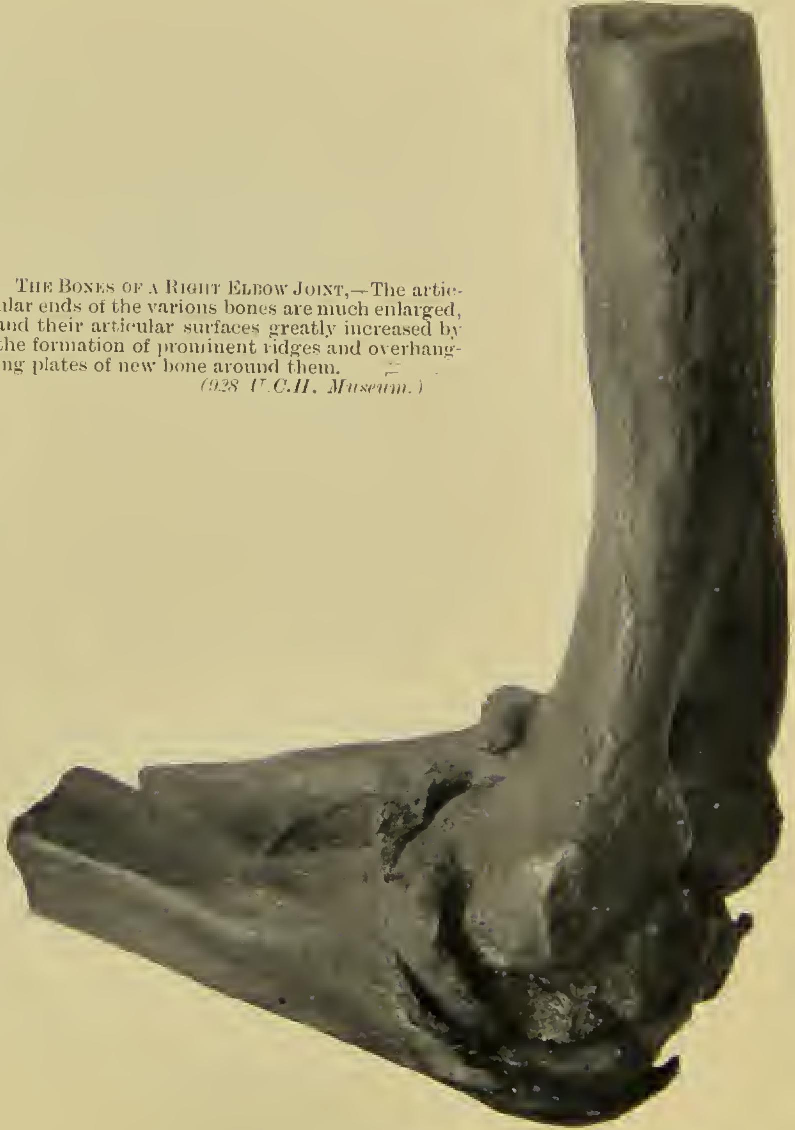
(972 U.C.H. Museum.)

PLATE XXII



A Right Scapula with the Upper Third of the Humerus.—The head of the humerus is encircled by a ring of new bone, the glenoid fossa is increased in extent and its surface rendered uneven by irregular patches of new bone which have been deposited upon it. The acromion process shows changes of a similar character.

(922 U.C.H. Museum.)



The Bones of a Right Elbow Joint.—The articular ends of the various bones are much enlarged, and their articular surfaces greatly increased by the formation of prominent ridges and overhanging plates of new bone around them.

(928 U.C.H. Museum.)

PLATE XXIII

A Section Forming a Right Hip Joint.—The superficial portion of the head of the femur has undergone absorption and its margins are encircled by a regular ring of new bone. The articular surface of the femur is much expanded and its surface rendered toothed or undulating, and into the depression produced by the destructive process new bony outgrowths from the acetabulum project. The margin of the acetabulum is studded by osteophytic outgrowths. The two surfaces are interlocked as by suture and the joint fixed thereby in an almost semi-flexed position. The bony tissue beneath the opposed surfaces has become very dense.

(958 U.C.H. Museum.)

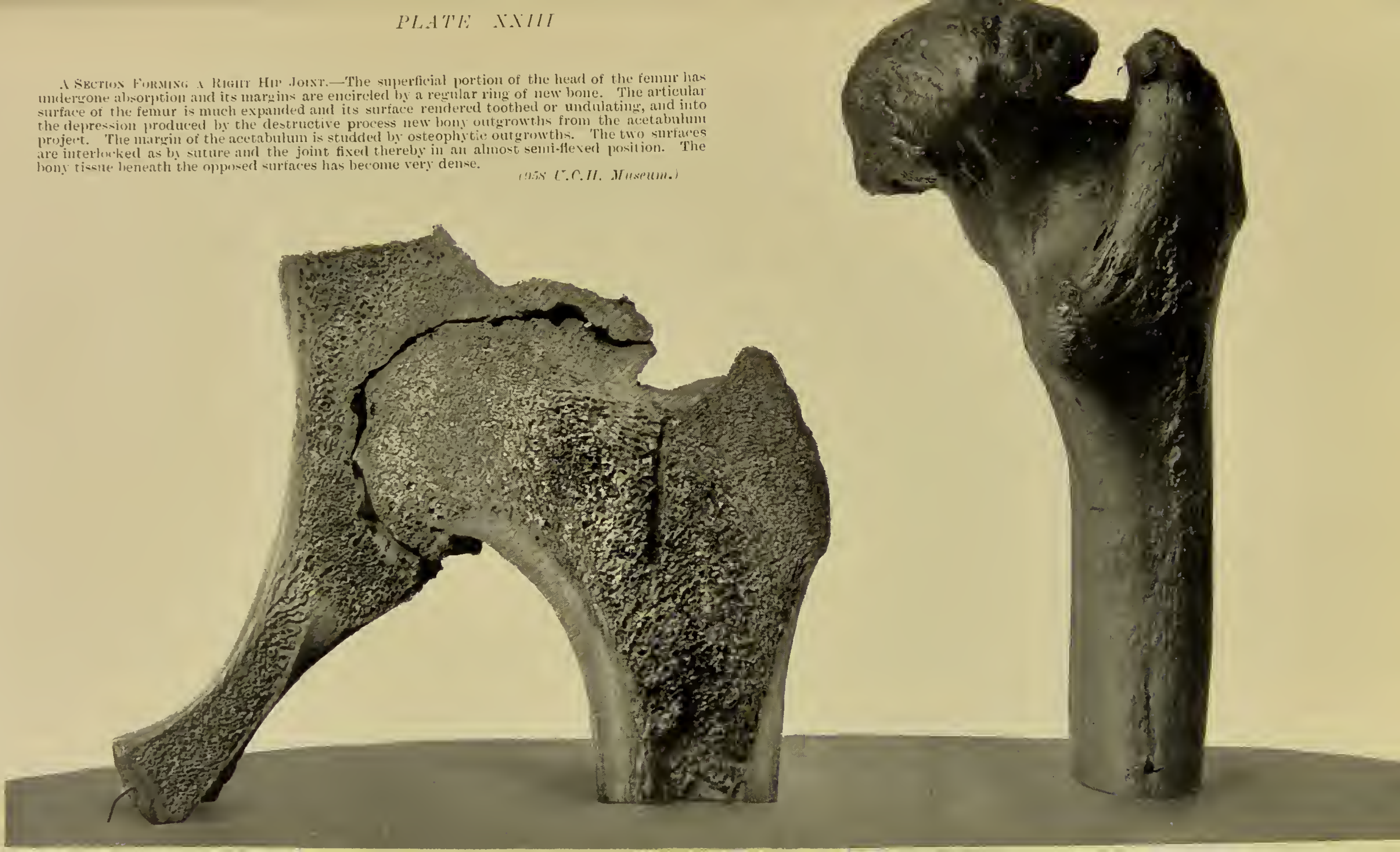
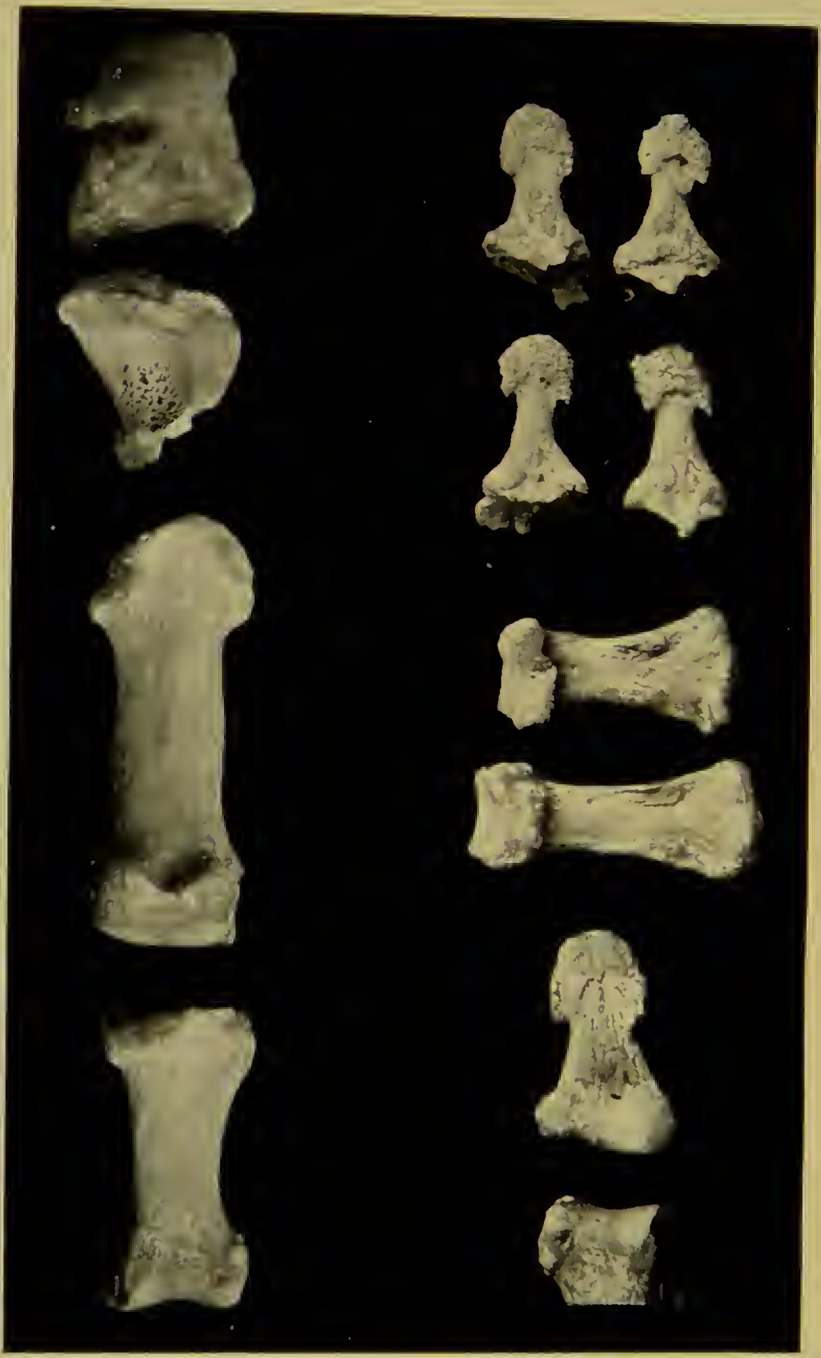


PLATE XXIV.



SOME OF THE BONES OF THE HAND, showing the effects of osteo-arthritis. The articular surfaces of the os magnum and semilunar bones are rendered irregular by ridges of new bone deposited around the articular surfaces; the latter show pitting and are in parts eburnated. The bones of the thumb show lipping of the articular surfaces, and similar changes are to be seen in the phalanges of the fingers.

(933 U.C.H. Museum.)

where pressure is less marked, the reverse takes place: the cartilage, instead of being thinned, becomes thickened, its proliferating elements forming irregular crests or nodules, which, primarily cartilaginous, eventually become ossified.

The osteophytic growths thus developed are the source of the deformity of the affected joint, and are responsible also for the limitation of its mobility, the restriction of which may, in isolated instances, amount to complete locking, but true bony ankylosis never occurs.

According to Ranvier[2] the localization of these bony formations more or less strictly to the peripheral portions of the cartilage is due to the fact that at this site it is covered by synovial membrane. This being so, the proliferating cartilage cells pent up beneath the enveloping membrane are unable, like those of the central portion, to escape into the articular cavity, and becoming heaped up, undergo chondrification and eventually ossification.

According to this authority, all these bony vegetations are derived from this source, but the periosteum probably participates, and in some examples the joint capsules, ligaments, and tendinous insertions show varying degrees of bony transformation.

The changes in the extremities of the bones next claim attention. Deprived of their encrusting cartilage, and exposed to unusual friction, their surface develops striking hardness and density, and presents a smooth porcelain-like appearance, in which a closer scrutiny reveals the presence of an infinite number of minute foramina, orifices of the Haversian canals.

Later on this usually thin layer of condensed bone shows parallel grooves along its surface which follow the line of direction of the movements of the joint, being very readily distinguished on the condylar and trochlear portions of the femur and the corresponding surface of the patella (*Plates XXI to XXIV*). Partly as the result of direct attrition, and partly owing to interstitial absorption, the neck of the humerus or femur becomes shortened, and the heads of these bones, flattened and encircled by bony asperities, present a truncated or crushed appearance. Being enlarged in this manner, the cavities for their reception must obviously become increased in extent; consequently the glenoid and acetabular recesses become enlarged and deepened, their rims studded with bony excrescences. To such an extent may this hollowing-out process proceed that the fundus of the acetabulum may bulge into the pelvic cavity, and as Adams[3] remarks, the bone in this situation under these circumstances may become exceedingly thin and almost diaphanous. The excavated fossa, deepened by the formation of bony excrescences along its rim, may indeed incarcerate the head of the femur, which, though readily movable, cannot be withdrawn from its socket, owing to its close embrace by the exostoses contracting the brim of the acetabulum. In addition to these modifications in the shape of the bones, the ligamentum teres is absorbed, and according to Adams the short fibrous bands which stretch across the notch in the acetabulum may become converted into bone. In the shoulder joint the intra-articular portion of the long tendon of the biceps may totally or partially disappear under the influence of the disease, and no trace of the glenoid ligament may be found in advanced cases. In addition the head of the bone, as the result of changes induced by its altered shape in the surrounding structures, may undergo displacement or luxation in various directions, such malpositions not infrequently, as Adams pointed out, having been confounded with the result of injury. Apart from these, the intra-articular fibro-cartilage of the temporo-maxillary joint, and according to Adams that of the acromio-clavicular articulation, may be utterly destroyed.

Passing now to the consideration of the shafts of the bones, these may be in no way altered, but on the other hand they may show a marked increase in their density owing to sclerosis of their central portions. Actual enlargements may also take place in various directions, and occasionally an increase in the length of the affected bone results. Adams relates a case in which, as the result of this disease, one ramus of the jaw was an inch longer than its fellow. As opposed to this hypertrophy and condensation of the osseous structures, atrophy and rarefaction of these same tissues may ensue when the disease attacks aged people. Cruveilhier long since described such an atrophied state in the structures of the temporo-maxillary articulation. Adams noticed a similar lightness and porosity in the bones of the shoulder joint, likewise of the carpus in elderly people. It is convenient here to refer to the fact that uratic deposits may co-exist with osteo-arthritic lesions, as was long since pointed out by Fuller,[4] and more recently Hilton Fagge[5] and Toogood[6] have confirmed his observations.

In some instances, for example, the metatarso-phalangeal joint of the great toe may show the combined lesions of gout and osteo-arthritis; in others this articulation may be the seat of gout, while the knees are the seat of osteo-arthritis; and in one of Hilton Fagge's cases, one knee joint was the site of osteo-arthritic changes, while the opposite articulation manifested uratic deposits.

The synovial membrane undergoes the following changes: it becomes more hyperæmic, while its villi undergo hypertrophy and fresh villous appendages make their appearance. According to Hoffa, who has made a special study of the minute alterations in the synovial membrane in osteo-arthritis, the following are the salient pathological alterations, in the production of which he distinguishes two stages:—

The first is characterized by villous overgrowth, proliferation of the fixed connective-tissue cells, with extensive pigmentary deposits. These latter are of a dark-brown hue and localized, partly extra- and in part intra-cellularly. Incidentally, new blood-vessels make their appearance in moderate number, with scanty perivascular leucocytic accumulations.

In the later stage of the disease, however, degenerative changes predominate.

The synovial membrane undergoes mucous and hyaline transformation, the ultimate and occasionally complete change of the synovialis into structureless hyaline tissue being considered by Hoffa to be characteristic of the affection.

The numerical increase and hypertrophy of the synovial fimbriæ may proceed to such an extent as to create a condition of chronic villous arthritis. Painter,[7] operating for the relief of symptoms of "locking" in the case of localized osteo-arthritis, subjected to histological examination one of the enlarged synovial tufts, a cross section of which revealed the following characteristics: the cortex was composed of fibrous tissue containing many small round cells, while the medulla consisted of connective tissue cells staining well in some places, in others showing no nuclei, leaving the fibres thin and separated.

Aggregations of fat cells occupy much of the remaining space. Round cells infiltrate the whole of the tissue, especially in and around the cortex, where they are arranged in definite nodules. Many vessels are scattered throughout, their walls thickened and in many cases obliterated. Sometimes cartilaginous bodies form in the substance of the hypertrophied fringes, which subsequently, through detachment of their pedicles, become free in the articular cavity, and occasionally undergo more or less complete bony transformation. According to Adams, during these initial stages of the disease an excess of synovial fluid is usually present in the joints. Later on, however, this redundant secretion undergoes absorption, and by this time the fibrous and synovial capsules of the articulation will be found to exhibit great thickening, these structures developing in some instances a remarkable degree of density.

Bearing in mind the important etiological rôle attached to trauma by most authorities in this disease, it is significant, as Hoffa remarks, that the anatomical lesions in this affection should so closely resemble those of traumatic arthritis.

As this observer suggests, it is quite possible that in many instances a long-forgotten trauma may have supplied the initial impulse to the morbid process. Such injury might readily determine the occurrence of a greater or less degree of hæmorrhagic intra-articular effusion, which, by its presence, would initiate what he terms a "reactive osteo-arthritis." It is easy to understand how, once started, the morbid process tends to perpetuate itself. Thus, during the natural exercise of a joint so affected, the roughened cartilaginous surfaces and bony asperities must be a source of constant irritation to the sensitive synovial membrane. This, in its turn, responds by renewed formation of connective tissue and hypertrophy of its tufts. These, once formed, contribute to the undue irritability of the joint, the elongated processes either becoming free in the cavity, or from time to time becoming incarcerated between the articular surfaces, and a vicious circle is thus established. This hypothesis as to the mode of action of trauma may possibly explain the excessive deposition of pigment noted by Hoffa as present in osteo-arthritis.

As before alluded to, we know from Bowlby's work that intra-articular hæmorrhages can initiate osteo-arthritic changes in the subjects of hæmophilia.

This sequence lends colour to the suggestion that in osteo-arthritis as the result of repeated "internal traumatisms" small entravasations of blood take place into the articular cavity, which, by their resorption, account for the presence of the unusual degree of pigment.

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CHAPTER XIII.

CLINICAL DESCRIPTION.

OSTEO-ARTHRITIS is essentially a disease of the middle and later periods of life. Rarely to be met with under forty years of age, the liability to the affection increases from this period onwards to the end of sixth decade, but not very infrequently its onset may be delayed until extreme old age, and this tardy appearance is especially characteristic of certain monarticular forms of the disease, such as malum coxæ senilis.

As opposed to this, however, isolated instances have been recorded of the occurrence of genuine osteo-arthritis even in young children. From the time of Adams it has been customary to recognize two types of the malady, a generalized and a localized form: the disease in the former implicating many joints, while in the latter its ravages are largely restricted to one articulation, for example, the hip joint. This process of differentiation reached its acme in the hands of Adams, who distinguished localized forms not only of all the larger, but also of some of the smaller, articulations, such as the temporo-maxillary. Nevertheless, as a matter of clinical experience one rarely if ever meets with a purely monarticular example of the affection, there being invariably some similar, though may be much less marked, articular lesion elsewhere. On the other hand, a widely generalized osteo-arthritis, involving not only the larger but the smaller joints of the extremities, is also not common, the tendency of the disease being to involve but a few joints, and these not symmetrically. In other words, the specific distribution is not poly- or monarticular but oligo-articular, and equally characteristic is its predilection for attacking the large joints. In both these respects therefore it contrasts with rheumatoid arthritis, which is polyarticular and symmetrical in distribution, with a distinct preference for the smaller joints. For the better convenience of description, however, these somewhat arbitrary subdivisions will be adhered to, it being fully understood that they are distinctly artificial and not sharply defined clinical entities.

It is proposed to deal first with the generalized variety, and afterwards to proceed to discuss in greater detail the particular features of the more localized types. When widely generalized the disease may, like rheumatoid arthritis, involve not only the larger articulations, but also the small joints of the hands and feet, together with the temporo-maxillary, and in addition some degree of implication of the spinal articulations may be present; but with this latter we shall deal more fully in the section devoted to spondylitis deformans. This widely-distributed form of osteo-arthritis is most commonly to be observed in women. As regards its mode of onset, in the majority of instances it is insidious in character, and the course pursued by the disease is slow, progressive, and for the most part apyrexial. Not infrequently the disorder is primarily installed in one of the larger articulations, and subsequently, after a variable period—it may be months or even years—an extension takes place to the other joints, the case ultimately assuming the picture of a generalized osteo-arthritis; or, on the other hand, the disease may in a comparatively short time involve to a mild degree several articulations, and subsequently the morbid process may, so to speak, centre itself in one of the larger joints, in which it progresses so rapidly and inveterately as to overshadow the similar affection of the other joints, where it pursues a more temperate course.

Nothing, however, that could be justly compared to the acute onset of some forms of rheumatoid arthritis ever takes place in osteo-arthritis, unless we include here those cases of acute infectious osteo-arthritis associated with specific micro-organisms, and upon which such stress has been laid by Nathan[1] and other observers. In these, however, the severity of the constitutional disturbance, taken together with the fact that the bony outgrowths are rather a sequence than a part of the disease—an effort, so to speak, at repair following the previous destructive inflammation—are sufficient to denote their distinctness from that form of osteo-arthritis under discussion.

Confusion with rheumatoid arthritis is most likely to arise when this generalized form of osteo-arthritis is encountered in young persons, and such cases being usually of the female sex, the possibility of error is of course thereby enhanced. Despite, however, a certain rough similarity, a little careful observation soon convinces one of the essential distinctness of the two affections.

To begin with, the subjects of osteo-arthritis, notwithstanding the severe and widespread arthritic mischief, are usually well nourished. Constitutional disturbance is slight or entirely wanting, the temperature is not raised, the pulse not quickened. In addition, pigmentation of the type so characteristic of rheumatoid arthritis is not to be observed, and there is usually a complete lack of any trophic disturbances in the skin of the extremities: no vasomotor phenomena, no localized sweatings occur, and not even in the most advanced cases do we see areas of glossy skin and so forth. Enlargement of the lymphatic glands is also wanting.

Again, sensory disturbances, a very prominent feature in rheumatoid arthritis, are much less so in osteo-arthritis. Pain in the latter may be almost absent, and even when complained of, careful enquiry will often elicit the fact that it partakes more of the nature of a stiffness or discomfort evoked by movement. In those instances in which persistent pain of a neuralgic character is present it will not infrequently be found to depend on osteophytic outgrowths giving rise to pressure on contiguous nerve trunks. Such neuralgic pains may follow the track of the sciatic, brachial, or intercostal nerves, and their incidence should immediately suggest the extension of the morbid process to the spinal articulations. It must, however, be borne in mind that such osteophytic outgrowths are not necessarily in intimate relationship with a joint; thus Goldthwait narrates that in one of his cases of osteo-arthritis obstinate pain in the heel was found to be due to a bony spur on the under-surface of the os calcis. In continuation a further point of distinction is to be found in the comparatively slight degree of muscular atrophy that obtains, and such as may be present is obviously of reflex arthritic origin, and unlike rheumatoid arthritis, even the suggestion that it might be of central origin has never been advanced. Some degree of muscular spasm may be present in the early stages, never, however, reaching that degree of intensity attained in rheumatoid arthritis.

It now remains for us to consider more in detail the external appearances presented by osteo-arthritic joints, and obviously the features will be more clearly defined in exposed articulations, as for example those of the fingers, more particularly the mid-phalangeal. Unlike rheumatoid arthritis when it affects these same articulations, the skin over the joints, as a rule, presents no abnormal changes, no semi-asphyxial or waxy tint prevails, and no localized perspiration on its surface is to be observed, and at no period, however advanced, of the disease does the skin undergo atrophic changes and assume a glossy or parchment-like appearance. Marked swelling of the joint, when present, is as a rule due to excess of synovial fluid and not to peri-articular thickening; thus we get no spindle-shaped appearance. On the contrary, the outline of the joint, instead of being smooth and rounded, is broken and irregular, its nodular contour being due to osteophytic outgrowths studding the line of junction of the bones forming the articulation. Such displacement of the bones from their natural direction as may occur is usually lateral, and due directly to the mechanical thrust of the bony excrescences, and is in no way related to muscular spasm or contracture. As opposed to this, in rheumatoid arthritis luxations and hyperextensions are the rule, for which in this affection muscular retractions are directly responsible.

This diversity between the articular lesions of rheumatoid arthritis and osteo-arthritis, though strikingly obvious even in the early stages of their development, tends to become more accentuated in the later periods of their life history. As we have shown elsewhere, the swollen one-time voluminous spindle joint of the former affection sooner or later undergoes a process of shrinkage, progressive atrophy overtaking all its constituent elements; skin, bone, and cartilage tending to wither away until eventually the articular structures dwindle into what may be termed the small end joint of rheumatoid arthritis. On the contrary, in osteo-arthritis, the more ancient and inveterate the lesions the greater the amount of new bony formations, the more pronounced the deformities produced, the more marked the enlargement of the affected articulations. In other words, while the destiny of a rheumatoid joint is, as a rule, to contract, even ultimately in some cases to become smaller than normal, that of the osteo-arthritic is to expand and increase, and this to a degree which in some of its more aggravated examples may fairly challenge comparison with the very similar changes met with in the nervous arthropathies. Again, when we come to consider the aspect of the hand as a whole when smitten with multiple osteo-arthritis, we note that in its general appearance and in the character and distribution of its lesions it differs widely from a rheumatoid hand. In the former the skin is of normal colour, and the hand feels warm to the grasp, with no abnormal moisture, while occasionally, as Garrod[2] has pointed out, the fingers may be the seat of diffuse brawny swelling. In the latter the skin is often paler than normal, or it may present a mottled appearance, the fingers are frequently ashen or semi-asphyxial in hue, while the affected member almost invariably strikes cold to the touch and is unpleasantly moist.

Again the terminal phalangeal joints, so rarely if ever enlarged in rheumatoid arthritis, are, on the contrary, the site of election for the most common of all osteo-arthritic lesions, Heberden's nodosities. As opposed to this, rheumatoid arthritis displays an equally marked affinity for the mid-phalangeal articulations, the spindle enlargement of which is so constant and distinctive a feature of the rheumatoid hand.

It is true that osteo-arthritis may also involve these same joints, but as Garrod points out, its marked preference is for the carpo-metacarpal joints of the thumb. A further point of contrast is afforded by the early and marked atrophy of the small muscles of the hand, the sunken interosseous spaces, and the flattened palmar eminences, features much less conspicuous in the osteo-arthritic member. With the progress of the disease these differences become still more pronounced: the osteo-arthritic hand with its enlarged and gnarled joints contrasting sharply with the shrunken and distorted appearance presented by that of the rheumatoid patient. In like manner all the joints of the upper limb—the wrist, elbow, and shoulder—are liable to become implicated in multiple osteo-arthritis, while in the lower limb the morbid process may attack the hip, knee, and ankle, as well as the articulations of the feet, including the toes. The particular characteristics of some of the most common and usually recognized local forms, such as those of the hip, knee, and terminal phalangeal joints of the hand, will be treated more in detail further on. In addition to the foregoing, Garrod considers that osteo-arthritis of the carpo-metacarpal joint of the thumb calls for special mention, as in instances—according to him not infrequent—these joints may be the only articulations which present signs of the disease, while in the majority of cases of the generalized forms these particular articulations are involved. While agreeing with this statement as to the frequency with which these joints are attacked in generalized osteo-arthritis, I cannot support this authority in his assertion that the cases in which these joints form the solitary sites of the disease are so numerous as to merit their designation as a special localized type of osteo-arthritis.

Because of its unusual character it is perhaps worth mentioning here that I recently saw an aged gentleman in which the disease was restricted to the metacarpo-phalangeal joints of both thumbs, and, as happens when the carpo-metacarpal joints of the same digits are affected, the utility of the hand was appreciably interfered with.

Again, as in rheumatoid arthritis, the temporo-maxillary joints may be implicated, though not so frequently. In such cases the affection tends to be symmetrical, but for long it may remain confined to one side, where it may pursue a much more inveterate course than in the opposite articulation. Patients afflicted with the disease in this site suffer considerably from pain of an aching character over the side of the face. It is aggravated by speaking and eating, and is sometimes referred to the ear. The creaking or crackling of the joint when in motion is very audible to the sufferer. On the other hand, stiffness rather than pain may be complained of, while increasing limitation in the possible excursions of the joint becomes manifest, and may, in severe and long-standing cases, interfere seriously with the capacity for eating, the patient having to resort to fluid nourishment transmitted through convenient gaps in the teeth. Adams,[3] in his vast experience, noted that deflection of the central point of the jaw might take place to one or other side when, as a result of the morbid process, the condyle and ramus of the maxilla on one side had undergone hypertrophy and elongation ; and in one of his examples, in which the disease was confined to the left temporo-maxillary articulation, facial palsy ensued upon the same side. I myself have seen cases in which advancement of the whole lower jaw had occurred as a result of symmetrical disease, the lower jaw protruding beyond the upper, a forecast of the usual malposition met with in old edentulous people. In conclusion, it is possible in some instances to feel below the zygoma an enlargement of the bone that passes down in front of the ear.

Similarly when the larger articulations of the upper extremities, such as the shoulder, elbow, and wrist, participate in the morbid changes, we get at the onset the same symptoms—pain or stiffness on movement, swelling from synovial effusion, and possibly some slight muscular atrophy, this latter especially noticeable when the shoulder is implicated. Occasionally the early intra-articular effusions, if excessive and persistent, give rise to a lax condition of the ligaments, with undue mobility of the joint ; such a complication is rare, the effusions tending to become quickly absorbed. On their subsidence the specific characters of the disease become more prominent : enlargement of the ends of the bones with osseous nodules, and excrescences skirting the edges of the articular surfaces, both readily appreciable by palpation and demonstrable by skiagraphy. As regards the deep reflexes, these usually, when the disease is of some standing, become more brisk, but this alteration in the reactions is more noticeable in the purely localized forms, when we have, moreover, the advantage of checking our results by those of the opposite and unaffected limb. Occasionally I have met with psoriasis in association with osteo-arthritis. According to Nathan the combination of this skin affection with mild osteo-arthritis is not infrequent.

It has been held that as a rule some degree of anæmia is present in the subjects of osteo-arthritis, and in order to ascertain the truth of this assertion, often based rather upon the personal appearance of the patient than upon actual blood examination, Erving[1] made a careful study of twenty cases of osteo-arthritis from this point of view. He tabulated his results as follows :—

Red blood corpuscles: highest 7,600,000, lowest 4,024,000; average 5,301,000; thirteen males average 5,640,000; seven females average 4,673,000. Hæmoglobin: highest 100 per cent, lowest 70 per cent; average 94·5 per cent. Leucocytes: highest 12,800, lowest 5,200; average, 9,175.

Leucocytes (differential):—Polymorphonuclear neutrophiles: highest 80 per cent, lowest 57·4 per cent; average 71·3 per cent. Small mononuclears: highest 3·50 per cent, lowest 14·4 per cent; average 21·8 per cent. Large mononuclears: highest 8·2 per cent, lowest 1·8 per cent; average 3·4 per cent. Transitionals: highest 4·6 per cent, lowest, absent in four cases; average 1·7 per cent. Eosinophiles: highest 5·6 per cent, lowest, absent one case; average 1·7 per cent. Basophiles: highest 1·2 per cent, lowest, absent thirteen cases; average 0·2 per cent.

No abnormal elements, such as microcytes, macrocytes, nucleated corpuscles, or other signs of hæmic degeneration, were to be found.

Consideration of the above data seems to bear out Goldthwait's opinion that the blood in osteo-arthritis shows no special change. We possess little or no knowledge as to any disturbances of metabolism which may occur in osteo-arthritis.

In Von Noorden's[5] recent work the following results of research along these lines are given:—

Johannessen, for example, as a result of an investigation into the metabolism of albumin in the case of a child with arthritis deformans, came to the conclusion that there were no serious departures from the normal. The patient retained nitrogen in large quantities, such irregularities in the nitrogen secretion as did obtain being adequately explained by the fluctuations to which the supply of nitrogen was liable. To this may be added that Drachmann and Stokvis, quoted by Pribram, have remarked a reduction in P_2O_5.

Von Noorden and Belgardt found a decrease in the calcium as well as in the phosphorus output. Daily retention was as follows: CaO 1·28 and ·750, MgO ·06 and ·034; P_2O_5 1·13 and 1·130.

In concluding this brief account of the signs and symptoms peculiar to osteo-arthritis, one cannot help being struck with the paucity of its collateral phenomena as compared with their abundance in rheumatoid arthritis. This, to our mind, constitutes the salient distinction between these two joint disorders: osteo-arthritis is much more a disease of joints, and joints only, than rheumatoid arthritis: in the latter the arthritic changes form only one feature of a very varied clinical picture, the distinctive facies of which is in large measure due to the associated phenomena above alluded to; indeed, lacking these, it would be in danger of losing its clinical identity.

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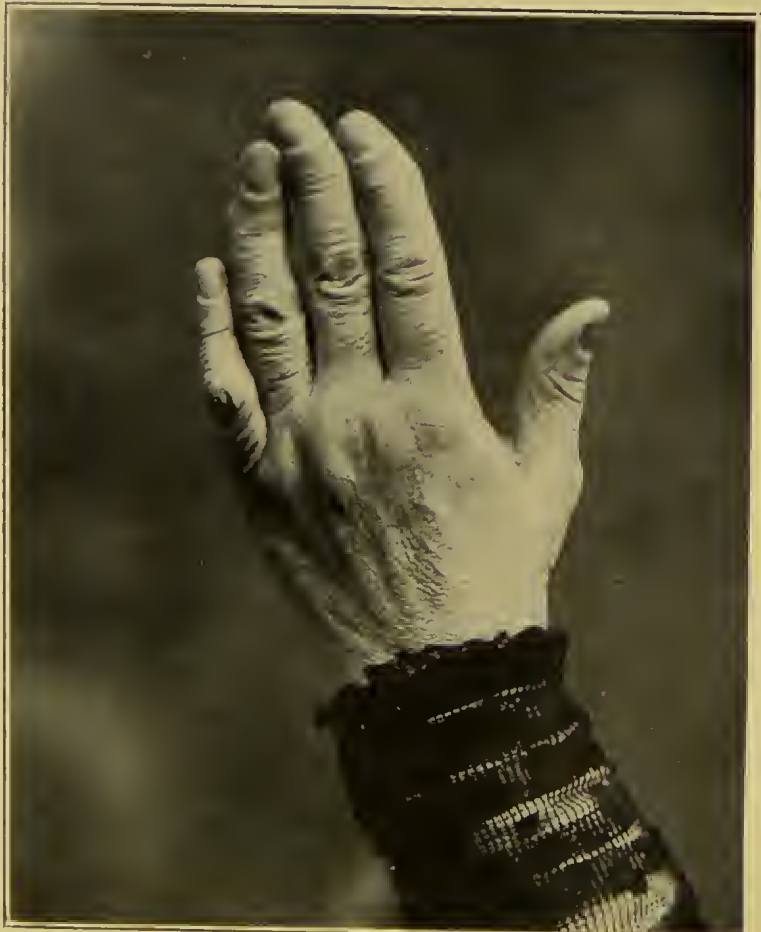
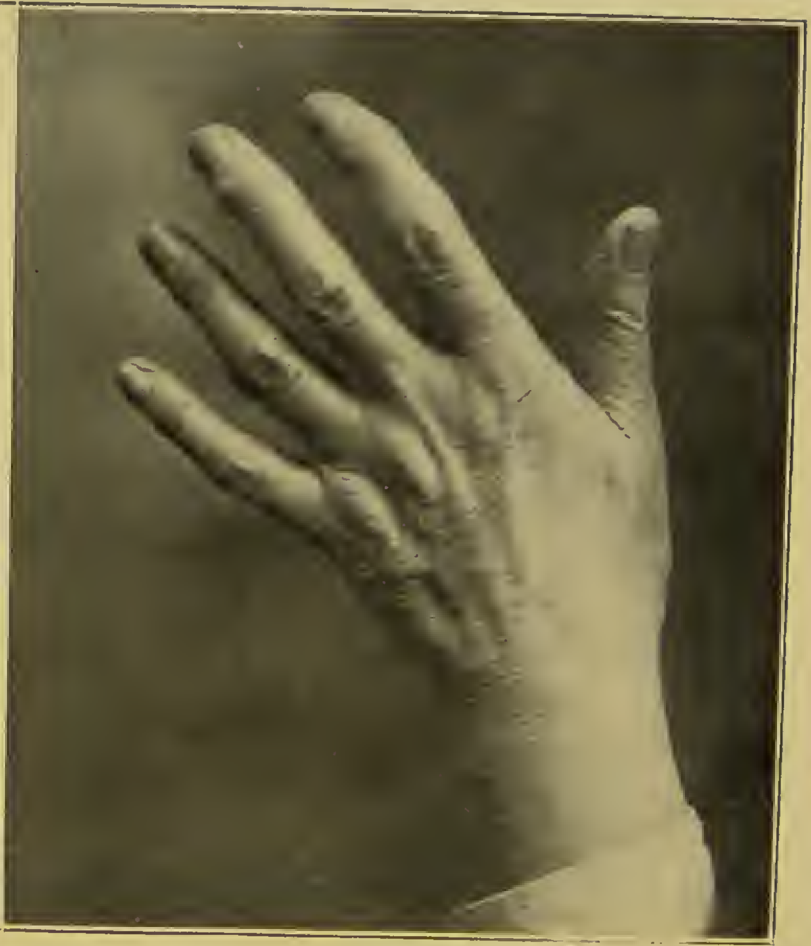
PLATE XXV.



Left Figure.—Osteo-arthritis of Hand, showing lateral deflection of terminal phalanges of index and middle fingers to ulnar, and of ring to radial side, by bony outgrowths. The patient was the subject also of long-standing osteo-arthritis of hip.



Right Figure.—Heberden's Nodosities situated on terminal phalanges of index and middle fingers.

Chapter XIV.

LOCALIZED VARIETIES.

HEBERDEN'S NODOSITIES.

The necessity of differentiating these particular manifestations of osteo-arthritis from gout was first realized by Heberden,[1] who, in his "Commentaries," asks the question, "What is the nature of these small hard nodules about the size of a pea so often met with in the fingers, usually a little above the extremities near the joints," and which, he adds, "have certainly nothing in common with gout, for they are found in many persons who have no experience of that disease ?"

Pathologically the anatomical lesions in this miniature joint disease are similar to those of osteo-arthritis of other articulations : fibrous thickening of the synovial membrane, erosion and absorption of cartilage, condensation and eburnation of the articular ends, with irregular osteophytic formations, leading to enlargement of the terminal phalangeal articulations and subluxation and deviation in the direction of the finger tips.

The onset of these enlargements takes place very insidiously, but occasionally their appearance is preceded by sensations of pricking in the fingers. In some instances attacks of redness, heat, and peri-articular swelling recur from time to time during the process of formation of the nodes. Painless as a rule, they give rise to little inconvenience, and are chiefly deplored on account of their unsightliness ; if, however, they are subjected to any trauma they develop much more quickly, and by their unusual size may seriously disable the use of the hand for more specialized occupations.

It is convenient here to refer to the fact observed by Goldthwait,[2] that in persons who already show Heberden's nodosities (*Plate XXV, right figure*) upon their terminal phalanges, osteo-arthritic changes may supervene in hitherto uninvolved joints as a result of apparently trivial injuries, and like him I am accustomed to warn such patients of the lamentable consequences that in their case may follow traumatisms, however slight. The nodes show a marked tendency to progressively involve all the end joints, including those of the thumb.

The lateral deflection of the terminal phalanges is due to the thrust of bony outgrowths, these being usually more pronounced on one or other side. According to Garrod,[3] in later stages of the disease the list of the terminal phalanges is toward the radial side. In many cases, however, adjacent finger tips may point in directly opposite drections (*Plate XXV, left figure*). There is usually also some degree of flexion of the end joints, but rarely, if ever, any hyperextension. The bony character of the enlargements is easily ascertainable by palpation, and can, if necessary, be confirmed by skiagraphy. There is usually some rigidity of the articulations, but no creaking as a rule. McCrae, however, has noted one instance in which definite crepitus was elicited.

It is commonly supposed that the nodes are but seldom accompanied by severe osteo-arthritic manifestations elsewhere, and while it is true that in some instances they may be the sole evidence of the disease, in my experience there is invariably some implication of other articulations, especially those of the mid-phalangeal or metacarpo-phalangeal joints. Spender,[4] who paid particular attention to this point, noted that in a series of 400 cases of the affection the most commonly associated lesions were situated in the first and second metacarpo-phalangeal joints. The relationship of the nodosities to gout has vexed physicians since the time of Heberden, but the majority now agree that there is no causal connection, though it is certain that the articular lesions of both conditions may coexist in the same individual, and according to Hilton Fagge[5] and others, in the same joint. Garrod even states that similar enlargements of the terminal joints may develop in gouty people, and this even in the absence of any definite uratic arthritis of the affected articulation, but he differentiates these from Heberden's nodosities by their asymmetrical distribution, their limitation to one or two end joints, and the more bulbous and less nodular appearance of the swellings produced.

Charcot,[6] as we know, considered that the nodosities presented affinities not only for gout but also for both forms of arthritis deformans, and more recently McCrae[7] has noted the "association of these lesions which we term 'osteo-arthritic' with those in other joints which are undoubtedly more of the 'rheumatoid' type." This coexistence of Heberden's nodes with rheumatoid arthritis he thinks certainly points to a possible association of the two types, a reversion in part to the view originally held by Charcot that Heberden's nodosities, general chronic rheumatism, and partial chronic rheumatism were but three forms of one and the same affection. My own opinion is that the nodosities are examples of osteo-arthritis, and that, although occasionally to be met with in association with rheumatoid arthritis, they are essentially as different and as distinct from that type of arthritis deformans as they are from gouty arthritis, despite the established fact of the occasional co-existence of the arthritic lesions of both gout and osteo-arthritis in one and the same individual.

MALUM COXÆ SENILIS.

(Syn. Senile Coxitis, Chronic Rheumatic Arthritis (Adams), Osteo-arthritis or Arthritis Deformans of the Hip.)

ETIOLOGY.

But little is known as to the etiology of this variety of localized osteo-arthritis. Poverty, damp, and exposure have been assigned as predisposing causes, but their presumed relationship admits of only a limited application, inasmuch as the affection is frequently met with in persons upon whom no such depressing influences have been at work. The onset of the malady not uncommonly dates from some injury, either trivial or severe, of the hip joint, and evidence pointing to the determining influence of trauma in the production of this and other forms of localized osteo-arthritis appears to be accumulating. In some instances the repeated performance of some special movement calculated to bring undue strain upon the hip joint seems capable by itself of initiating the disorder; thus, for example, in a well-marked case under my own observation the patient, an elderly lady, attributed the beginning of her trouble to the oft-recurring "strains" experienced by her in mounting an unusually high dogcart. In other cases, as Adams[8] noted, the onset of the affection is apparently determined by direct violence, more especially by severe blows or falls upon the great trochanter. Such indeed appeared to be the probable explanation of an example of the malady which came under my notice recently, in which the patient's left hip became affected subsequently to a hunting accident, in the course of which the great trochanter on the affected side sustained a severe concussion owing to the rider's horse falling heavily over on to his left thigh. In this connection it is important to remember that the clinical appearances presented by osteo-arthritis of the hip, in its more advanced stages, may closely simulate those met with in an impacted fracture of the neck of the femur, and when we bear in mind the insidious onset of the disease it is easy to see how readily an error in diagnosis may take place in such a case when an intercurrent injury involving the joint has occurred.

Adams long since pointed out the possibility of confusion in this direction, citing the well-known case of Mathews, the celebrated comedian, in proof of his contention that not only might the clinical features of the two conditions be confounded during life, but also their associated morbid changes when disclosed at autopsy.

Briefly considered, the salient features of this eminent actor's case were as follows: While driving he was thrown from his gig, and sustained a severe injury to his hip, which resulted in permanent lameness. Notwithstanding that he was seen by some of the most eminent surgeons in London of that time, it was found impossible to decide as to the presence or absence of a fracture. After his death, ten years subsequently, the affected hip-joint was removed and exhibited by Mr. Snow Harris at a meeting of the Medical Association, the morbid changes present in the articulation being supposed to settle in the affirmative the vexed question as to whether or not bony union of an intra-capsular fracture of the neck of the femur could take place.

Among those present on this occasion was Adams, who, after scrutinizing the specimen, pronounced the anatomical changes to be due to "chronic rheumatic arthritis," an opinion to which he had the satisfaction of converting most of those present, including the exhibitor.

That Adams's warning of this possibility of confusion with fracture was well founded, may be gathered from the following passage, quoted from Lovett and Bradford's well-known work on orthopædic surgery, in which they have devoted a section to "arthritis deformans of the hip joint." These authors[9] state: "The position which the limb assumes in the more advanced cases of the disease is one which is calculated to be most misleading, especially when the affection has followed a fall upon the great trochanter. The limb may be rotated outward, and with the apparent shortening presents almost a complete picture of an impacted fracture of the neck of the femur." Apart from injury, as Garrod points out, an osteo-arthritis may develop in a hip joint which in early life was the seat of disease long since quiescent.

Recently George Preiser,[10] of Hamburg, has advanced the novel view that "arthritis deformans of the hip" depends upon an abnormal position of the acetabulum, the cotyloid cavity being situated either too far forwards or too far backwards, this malposition leading to limitation of outward or inward rotation respectively. In other words, he contends that the disease of the hip is secondary to the "statical incongruity" just indicated. The variety in which the cotyloid cavity is located too far forwards is chiefly seen in association with rachitic pelves, but the author is unable to assign any such etiological cause for the second variety, in which the acetabulum is situated too far posteriorly. The author found one or other of these malpositions present in twenty-four cases of the affection, and he states that in all cases of arthritis deformans of the hip the trochanter is above Nélaton's line, and this not only on the affected, but also on the unaffected side, thus supporting his claim that the hip condition is secondary.

The affection displays a marked predilection for the male sex, though it occurs occasionally in women, and has been met with even in children. At one time it was thought to be confined to periods of advanced life: hence the term "malum coxæ senilis" applied to it by Adams, who, however, having in the course of a more extended experience met with many instances of its occurrence in persons between thirty and forty years of age, subsequently designated it "chronic rheumatic arthritis of the hip joint," inasmuch as neither the age incidence nor the associated morbid structural changes, to his mind, justified any distinction being drawn between it and "chronic rheumatic arthritis" of other articulations. It still remains true, however, that the majority of the cases occur in males over fifty years of age. As regards its occurrence in children, George Preiser, in his recent monograph on the subject, describes an example of "idiopathic juvenile arthritis deformans coxæ," and notes that fourteen instances of its incidence in children have been recorded by various German observers. Lovett and Bradford also state that it may occur in children. As a rule the disease affects only one hip, but Adams and Charcot[11] have related examples in which both hip joints were implicated, and Garrod[12] considers that a slight involvement of the opposite hip is not uncommon. Not uncommonly, when the disease has been strictly localized to the hip joint for many years, an extension of the osteo-arthritic process to other articulations takes place. In a case now under my care, the knees have become involved secondarily to the left hip joint, while the terminal phalanges have developed well-marked Heberden's nodosities. Much more rarely, as Teissier[13] remarks, the reverse sequence occurs, the hip joint only becoming implicated after osteo-arthritic changes have been present for a variable period in other articulations.

CLINICAL FEATURES.

The onset of the affection is usually insidious and its progress slow, the patient as a rule complaining only of slight pain or stiffness in or about the hip joint. The stiffness is more marked in the early morning, wearing off with exercise during the course of the day, only, however, to return again at night, in greater or less degree according to the amount of exertion undergone. Pain soon becomes a prominent symptom, and in the later stages is almost constant, save when the patient is completely at rest. Besides being felt in the hip joint it may be referred to the groin, and front of the thigh as far as the knee, and commonly it follows the track of a sciatic neuralgia. Lovett and Bradford indeed contrast the paths of pain in osteo-arthritis and tubercular disease of the hip in that the pain in the former often runs down the back of the thigh instead of down the front as it does in the latter disease.

A slight limp soon becomes noticeable, and the patient, otherwise often in excellent health, finds to his annoyance that his activity is becoming curtailed in various ways. Thus, in an acquaintance of mine whose left hip was thus affected, one of the first symptoms to attract his notice was the increasing difficulty he experienced in mounting his horse, the increasing limitation in the forward flexion of his left thigh ultimately condemning him to the necessary humiliation of a mounting-block.

Similarly, these patients, when forward flexion of the thigh is interfered with, find, when they attempt to sit down, that they cannot as formerly sink with ease into the depths of a chair, but are compelled to lower themselves gently on to the extreme edge of the seat, keeping the affected thigh as far as possible in a direct line with the axis of the trunk, with the leg either remaining fully extended or flexed at the knee. As might be expected with such limitation, crossing the affected leg over the opposite member may be absolutely negatived, or if possible, can only be achieved with great pain and difficulty, the aid of the hands often being enlisted to raise the crippled limb into the required position. On the contrary, it may be that the patient can only find ease from suffering when the affected limb is crossed over the other. Adams, in his work, tells us that this was the case in the instance of Perceval, a Dublin physician, who had suffered for many years with osteo-arthritis of the right hip. Perceval, having during his lifetime expressed a wish that his hip joint should be subjected to post-mortem examination, Colles performed an autopsy, during which he ascertained the presence of a bony outgrowth in front of the right acetabulum, over which the anterior crural nerve, " flattened and redder than natural," passed, this circumstance presumably accounting, in Colles's opinion, for the intensity of the patient's sufferings, and the freedom from pain experienced when the right leg was crossed over the left.

In the more advanced stages of the malady the clinical picture presented is very characteristic. When standing, the patient, usually an elderly man, leans heavily on a stick, holding his body a little backward, while he throws his weight as far as possible on to the sound leg. The affected limb is carried a little forwards, and in a position of slight adduction, with the foot everted, the heel often being slightly raised from the ground. The limb has the appearance of being considerably shorter than its fellow, but this is more apparent than real, being largely due to the obliquity of the pelvis, the spine of the ilium on the affected side showing considerable elevation. On the other hand, according to Lovett and Bradford, in the earlier stages in some instances abduction and apparent lengthening of the limb is met with, as in hip disease. In cases of long standing, however, real shortening may be present, owing to absorption of the head of the femur. A secondary curvature of the lumbar column with its convexity to the affected side is sometimes to be noted.

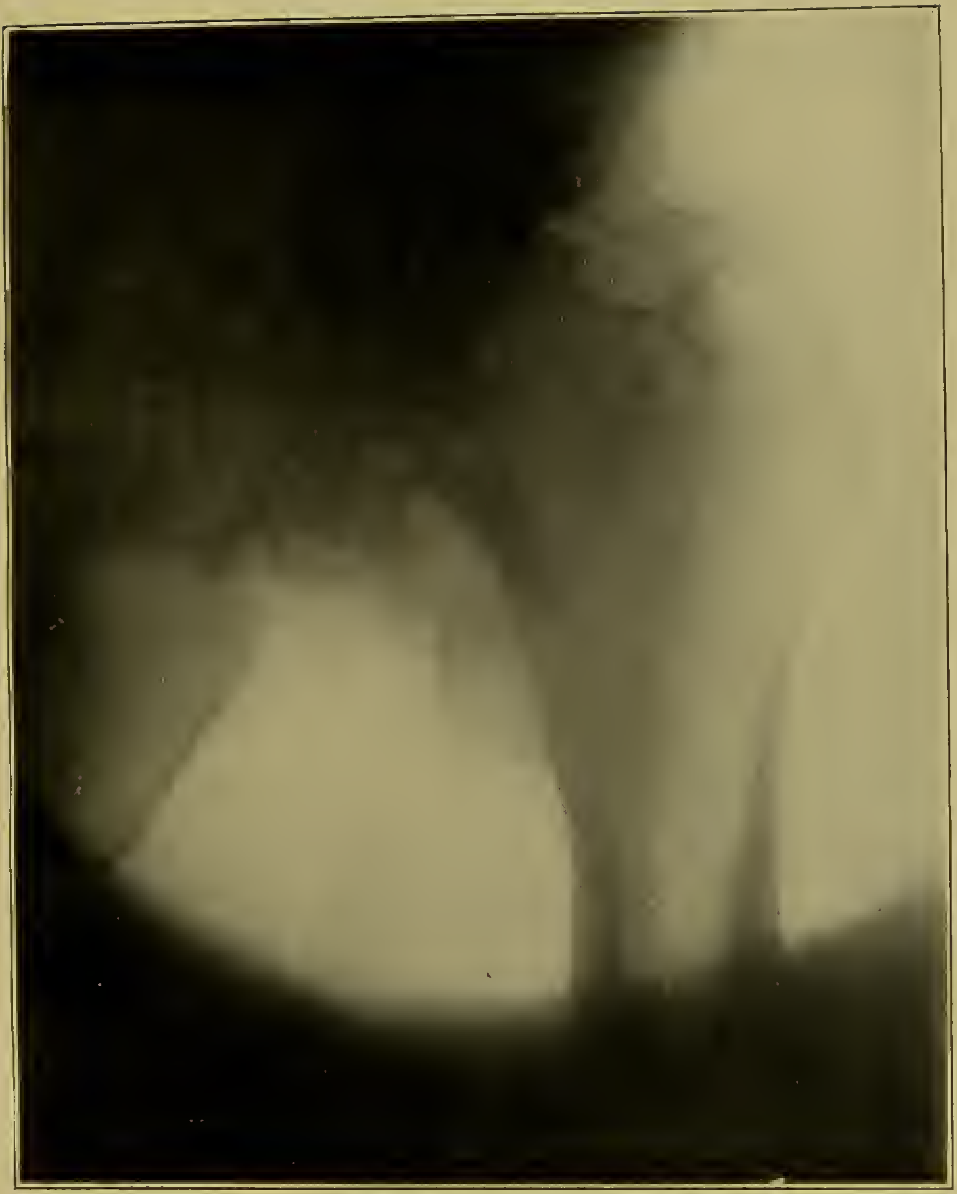
Wasting of the gluteal and thigh muscles takes place, but the calf muscles are undiminished in size. The knee-jerk of the affected limb is usually exaggerated. Examination of the patient in the horizontal posture reveals the presence of marked limitation in the movements of the hip joint, especially as regards abduction and external rotation, and in advanced stages fixation of the joint in a position of flexion or adduction occurs, or, as Lovett and Bradford point out, it may become rigid in a normal position. Actual ankylosis, however, does not occur.

Movements of the hip, whether passive or voluntary, give rise to creaking or coarse grating, often audible to bystanders. Palpation frequently shows the trochanter to be prominent and enlarged, and the presence of exostoses in the region of the acetabulum may sometimes be established.

The cysts described by Morrant Baker[14] may occasionally be found in connection with the diseased joint. These may reach a considerable size, and the tension of their contents may be such that the fluid escapes into the surrounding tissues.

In one case under my own observation, in which both hips, as well as the knees, were the seat of osteo-arthritis, large cysts, apparently in connection with both hip joints, were present. The outlook in these cases is gloomy, inasmuch as the morbid changes tend to progress despite all modes of treatment.

PLATE XXVI.



Skiagraph of Osteo-arthritis of Hip.—Showing absorption of the neck, with mushroom-shaped expansion of the head of the bone, by osteophytic outgrowths. New bony deposits in the neighbourhood of summit of the great trochanter.

DIAGNOSIS.

Excluding the possibility, more remote since the introduction of skiagraphy, of confusion with the results of fractures of the neck of the femur, the conditions most likely to be confounded with malum coxæ senilis are tubercular disease of the hip, and sciatica.

In tubercular disease of this articulation the subject is usually young, the general health is often deteriorated, and there may be signs of tubercle elsewhere. Locally there is greater tenderness on manipulation and more general fullness and swelling of the joint, the buttock shows no flattening, the muscles of the leg, as well as the thigh, are wasted, and the X-ray appearances of osteo-arthritis are absent.

In sciatica the pain is more constant, and is often just as severe at night when the patient is lying down as when he is up and about.

On the other hand, in osteo-arthritis, although the sciatic pain may be almost continuous during standing or walking, it usually subsides when the recumbent position is adopted, only disturbing the patient's rest when he attempts to alter his position. Wasting of the muscles when present is more generalized in sciatica than in osteo-arthritis, the atrophy in the latter being confined to the gluteal and thigh muscles. The gluteal muscles not being supplied by the sciatic trunk, any atrophy of them in the subject of sciatic pain should direct attention to the hip joint.

The degree of limitation in the movements of the hip joint in sciatica is determined by the amount of tension brought to bear upon the nerve trunk; thus flexion of the thigh and subsequent extension of the leg is intolerable, but hyper-extension of the thigh is permissible, also abduction and external rotation. In osteo-arthritis the limitation in the range is more general, and the movements of abduction, external rotation, and hyper-extension may be just as painful and restricted as those of flexion.

The differential diagnosis of these two affections may be very difficult in some cases, and in such skiagraphy (*Plate XXVI*) should be resorted to for the purpose of excluding or establishing the presence of osteo-arthritis, inasmuch as the two conditions may coexist.

OSTEO-ARTHRITIS OF THE KNEES.

This chronic affection, so commonly met with in women at the menopause, arises in the vast majority of instances very insidiously. Being rather of the nature of an infirmity than a disease it is apt to be disregarded or dismissed as "rheumatism," or may be construed as a sign of oncoming age, until the persistent and increasing disablement induced by the condition provokes alarm and compels attention. This indifference is hardly to be wondered at when we consider how seldom actual pain in the knees is complained of. In those few instances in which it is present it is referred to the inner condyles of femur and tibia, or to the under-surface of the patella, and but rarely, if ever, at all events in the early stages, is it severe enough to prevent walking. Stiffness, on the other hand, is a source of great discomfort, and constitutes the chief, and often the only, reason for which those patients seek advice. It is most marked in the morning hours, is ameliorated by exercise, but recurs obstinately after sitting for any period of time, and such patients, by design, pick out the loftiest chairs in their neighbourhood, so as to diminish the effort required in rising.

For the same reason going up and down stairs, especially the latter, worries them not a little. Exposure to cold and damp undoubtedly aggravates the stiffness, and the disease tends to progress more rapidly also in persons who by reason of some special occupation are compelled to kneel frequently, such as domestic servants, dressmakers, etc. In some instances these patients complain that when walking they occasionally experience a momentary sensation of uneasiness pointing to something having got between the bones of the articulation, but these symptoms of "locking" are usually mild in character, though they may in isolated cases be of marked intensity. Adams tells of a man the subject of osteo-arthritis of the knees in whom this intrusion of a foreign body invariably gave rise to excruciating pain, and a sudden fall in consequence. In quite early stages examination may reveal nothing abnormal in the appearance of the knee joints, swelling may be completely absent, and no irregular bony outgrowths may be detected, but despite this absence of any apparent alteration, if the palm of the hand be applied with firm pressure to the patella, and it be moved laterally over the condylar surfaces, a lack of smoothness in one or both of the opposing surfaces can be readily appreciated, and this may become audible as a creaking or grating sound, when the joint is alternately flexed and extended.

In other cases more acute in character there may be well-marked signs of effusion into the synovial sac. All the surface depressions are wiped out, and where the distention is very marked, the bursa beneath the inner head of the gastrocnemius may show plainly in the popliteal space when the limb is extended, but as usual disappearing during its flexion.

Again, similar effusions, though of a more transient type, may recur again and again in the course of the more insidious and subacute forms, when the joint has been subjected to injury, and such need not necessarily be of extraneous origin, but may arise in sequence to internal traumatisms from projecting bony spurs or foreign bodies interfering with the normal mechanism of the articulation. In the more advanced stages of the affection the effusions tend to be absorbed, and the characteristic irregular bony deformities of the joint become more obvious, when it will be noticed that the patella is increased in width, while the edges of its trochlear surface are studded with bony crests, and in addition osseous deposits may be felt along the margins of the femoral condyles and around the articular edges of the tibia, and may be at the insertion of the ligamentum patellæ. In the more advanced stages the knee joint, looked at as a whole, is often inclined inwards : the bones of the legs are usually rotated on their long axis outwards, with consequent eversion of the foot.

In cases of very long standing a greater or less degree of permanent flexion of the knee ensues, and the patella may be displaced outwards so as to lie on the outer condyle of the femur, or even beyond it, while the tibia may be displaced outwards and backwards towards the popliteal space.

It is remarkable, however, that notwithstanding the grave disorder of the joint, muscular atrophy is comparatively slight and is generally restricted to the extensors of the knee. When tested electrically, these muscles, more especially the belly of the vastus internus, may show delayed reaction time as compared with the musculature of the rest of the limb. The consequent over-action of their antagonists the hamstrings leads to muscular imbalance, and this appearing early, as it may in the disease, before limitation of the movement of the joint by bony deformities occurs, presumably explains the stiffness complained of so bitterly in the initial stages of the affection. It may not perhaps be out of place to emphasize the fact that the recurring and synovial effusions are not infrequent during the course of osteo-arthritis of the knees, their occurrence being indicative usually of the presence of villous outgrowths or bony spurs.

Otherwise the case, if first seen during the course of an effusion, may be treated as an ordinary synovitis of idiopathic or traumatic origin, the underlying morbid changes being overlooked by reason of the difficulty of eliciting grating, or palpating new bony formations.

The natural tendency of the disease, if untreated, is to progress, but the encouraging results of treatment in the early stages fully warrant one in laying stress upon the value of early recognition of the true nature of the arthritic trouble, which, if neglected, may make walking, always difficult, eventually impossible.

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CHAPTER XV.

DIAGNOSIS AND PROGNOSIS.

DIAGNOSIS.

THE conditions likely to be confused with osteo-arthritis are those of rheumatoid arthritis, chronic gout, and the nervous arthropathies.

Summarizing the chief characteristics of osteo-arthritis: It is a disease rarely met with under forty years of age. The mode of onset is generally insidious, never really acute. It attacks both sexes equally. Although it may be polyarticular, its specific tendency is to be mono- or more accurately speaking oligo-articular, with no marked leaning towards symmetrical distribution. It displays a marked predilection for attacking the large joints, such as the hip and shoulder.

Constitutional symptoms, such as emaciation, rapid pulse, pyrexia, pigmentation, and trophic disturbances, are generally absent. Muscular atrophy is slight, hardly ever pronounced, likewise muscular spasm and contracture.

Local characters of the articular swelling: general swelling, if present, due to synovial effusion, not to peri-articular thickening: hence no spindle-shaped appearance. Joint abrupt and nodular in shape. Displacement of the bones, if present, lateral in direction. The older and more inveterate the articular lesion, the greater the enlargement of the joint.

Rheumatoid Arthritis.—This affection differs from the foregoing in that it is most commonly met with in persons under forty years of age. It attacks women much more frequently than men. Its specific distribution is polyarticular, with marked leaning towards symmetrical invasion of joints. It evinces a pronounced preference for the smaller joints, the hip and shoulder often remaining immune.

Constitutional symptoms are much more conspicuous, pulse often quickened, temperature in more acute forms raised, extreme emaciation not uncommon. Muscular spasm prominent, muscular atrophy intense and widespread, with contractures. Trophic disturbances of varied nature and degree always present, such as pigmentation, vasomotor phenomena, and in advanced stages glossy skin.

Local characters of articular swelling: skin over joint waxy-white or semi-asphyxial in tint. Outline of joint smooth, rounded and spindle in shape, with, as a rule, no irregular bony projections at the line of articulation.

Deformities are due to muscular spasm, and are usually in the direction of luxation and hyper-extension, but rarely if ever is there lateral displacement.

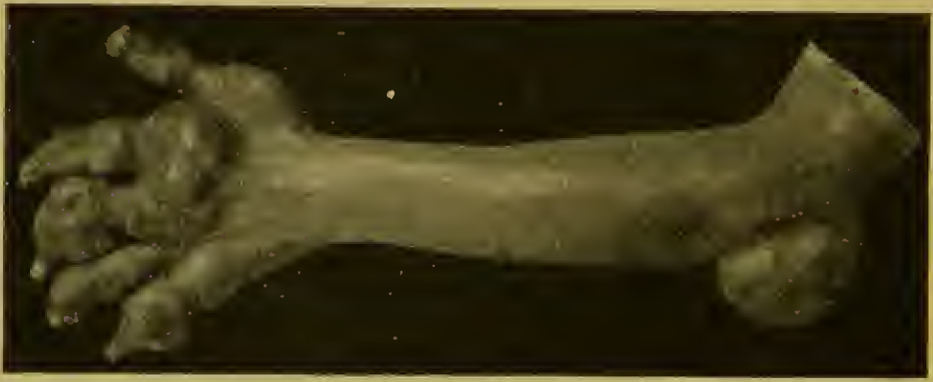

Fig. 1.—Wax Model of Left Hand and Forearm of a gouty patient. Large tuberous swellings due to uratic deposits are to be seen over the hand and fingers and at back of elbow joint. *(991 U.C.H. Museum).*

Chronic Gout.—The points to be remembered in differentiating this condition are as follows:—

The family history must be investigated for evidences of ancestral gout, likewise personal history, noting the occupation and mode of living, especially in regard to the use of alcoholic beverages. A history of previous acute attacks in the joints of the great toe, with the presence of tophi in the ears and elsewhere, will furnish direct proof of a gouty taint.

It is in the case of women past the age of the menopause that difficulties are most likely to arise. Although, as a rule, up to that period exempt from acute arthritic manifestations of that nature, the female sex not uncommonly at this epoch manifests symptoms of irregular gout. Moreover, such symptoms may be engrafted upon a previously existing osteo-arthritis, and this statement as to the co-mingling of the two conditions, as we have shown, rests not only upon clinical data but upon pathological proof of the co-existence of the lesions of both diseases in the joints of one and the same individual.

Arthropathies of Nervous Origin.—These remarkable joint affections, whether of tabetic or syringomyelic origin, are distinguished by their sudden fulminant onset, as a rule without premonitory symptoms. The joints become the seat of rapid and excessive intra-articular effusion. Tenderness, heat, and—more particularly—pain are completely absent.

In tabes the large joints of the lower extremities, in syringomyelia those of the upper, are the sites of predilection. In addition, in both diseases, associated phenomena pointing to extensive spinal disease will be present, such as ataxy, pupillary changes, absence of knee-jerks, and lightning pains in tabes; in syringomyelia dissociated anæsthesia, trophic disturbances of bones, progressive muscular atrophy, with paralysis.

PROGNOSIS.

The eroded cartilages being destitute of any powers of reproduction, and the actual alterations in the osseous structures being inevitably, at all stages of their development, permanent and fixed, it is obvious that *restitutio ad integrum* is unattainable. Nevertheless, by appropriate treatment in its early stages, the disease can, in many instances, be arrested, and the subject thereof enabled to follow a life of comparative usefulness.

Such desirable results are naturally more often obtained in the case of younger people. Even where the disease is generalized, the prognosis is much more favourable than in rheumatoid arthritis, owing to the comparative absence of muscular contracture with its associated crippledom.

Obviously, the older the patient, the more sombre the prospect, and in the presence of arteriosclerosis at any age, the arthritic mischief seems to pursue a more relentless course.

In the senile affection of the hip-joint, little, of course, can be expected; but, as Garrod has remarked, in that form of osteo-arthritis which attacks the knee of women at the menopause, the results of early treatment are distinctly encouraging—

sufficiently so, in my opinion, to warrant a more favourable prognosis being given than in any other variety of the disease. In conclusion, according to Teissier, the most frequent terminal complication is albuminuria. Hitherto, this statement lacks confirmation; but no one will dispute the general truth of Haygarth's reflection that "this is a disease which sadly embitters, but does not shorten the duration of, life."

CHAPTER XVI.

TREATMENT.

NOTWITHSTANDING the unalterable character of the bony and cartilaginous lesions in this disease, its treatment is, as Garrod[1] says, by no means as hopeless as some would lead us to believe. Goldthwait,[2] for example, considers internal treatment to be of little avail except in so far as special symptoms or defective elimination call for attention. My own experience is that internal treatment, both medicinal and dietetic, does exert considerable influence in ameliorating the symptoms and arresting the further progress of the malady.

DIETETIC.

In the more general forms of the disease no restriction or particular modification of diet is called for, and the patient should take abundance of good nourishing food.

On the other hand, in the localized forms, more especially that form of osteo-arthritis in the knees which attacks women at the menopause, modification of diet is often called for. The onset of the disease at this period frequently coincides with a notable increase in the patient's body weight, and in my opinion this, through its mechanical action, promotes the advance of the disease. It must be remembered that in this affection, as opposed to rheumatoid arthritis, bony nodes are of constant occurrence, and by reason of their presence the joint is in an extremely irritable condition in the early stages of the malady. Any notable increase in the patient's weight, therefore, is calculated to aggravate the irritation produced by these bony outgrowths. Goldthwait[2] and Painter[3] have shown that localized villous arthritis, not to mention recurring synovitis, may result in these cases from irritation caused by bony spurs.

In this particular type of the disease, I have found it of the greatest value to reduce, or even abolish for a time, the carbohydrate and fatty constituents of the diet, and also all malt liquors. Indeed, alcohol, as far as possible, should be rigidly excluded, or only a very moderate quantity of good whisky, or a light wine—such as Moselle—should be permitted. All forms of meat, poultry, fish, and game may be partaken of freely. All vegetables, also, with the exception of the roots and tubers such as potatoes, etc., may be eaten. Bread should be largely reduced in quantity, and only eaten after being thoroughly "torrified." Sugar should be prohibited, and saccharin, or saxin, used as a sweetening agent. Milk, cream, and butter should be very strictly limited. Hot water may with advantage be taken either an hour before or two hours after meals for its flushing effects. Should the urine become concentrated and contain a large amount of urates while on this proteid diet, barley-water or some alkaline mineral water should be freely partaken of. At meal-times fluid should be strictly limited.

From an extensive experience of this class of case I have no hesitation in saying that a restricted dietary forms a very valuable and essential portion of their treatment, and it fully deserves a trial in all cases in which the disease is localized to the lower extremities and is attended by more or less obesity. It must be remembered also, that in women at the menopause irregular manifestations of gout are prone to appear, and when these present themselves the diet must be modified accordingly. With these exceptions the diet should, as in rheumatoid arthritis, be of nutritious and varied character.

TREATMENT BY DRUGS.

Among the drugs recommended for this disease may be mentioned guaiacum, sulphur, arsenic, and the iodides, and to these may be added aspirin. In my own experience, the most useful and reliable for routine treatment are guaiacum and sulphur given either in tabloid form or in a cachet. The usual dose of 3 gr. of each is best taken immediately after meals three times a day. In order to obtain the most beneficial results from these agents, they must be given in doses adequate to produce a mild purgative effect. In many instances the above doses are not sufficient, but it will rarely be found necessary to exceed 15 gr. of each per diem. I know of no combination of drugs which exerts so marked an effect in diminishing, or removing, the muscular stiffness of which these patients complain so much.

Arsenic, as in rheumatoid arthritis, is most serviceable in certain cases. It is especially indicated when a dry and inactive state of the skin is present, and it may be added to the above combination in the form of arsenious acid or arseniate of soda. It is not advisable to push it to the degree of producing toxic effects, and its use should be intermitted from time to time.

Where marked swelling and effusion are present, iodide of sodium may be given in combination with guaiacum and sulphur. In cases arising at the menopause the protean symptoms incidental to that period are likely to be superadded, and for their amelioration nothing is superior to the preparations of valerian, which may be added to the drugs already mentioned.

While in the localized forms of the disease guaiacum and sulphur are more useful for routine medicinal treatment, in the generalized forms it is better to rely on the combination of arsenic and syr. ferri iodidi, to which may be added cod-liver oil, and these drugs must be persevered with for a long time.

For the relief of pain in all forms aspirin is the most reliable drug, and, as far as I have been able to ascertain, no ill effects follow its prolonged administration. When it disagrees, it does so almost immediately, causing sickness, which necessitates its abandonment.

LOCAL TREATMENT.

The most serviceable application we possess is the iodide and soap liniment, a small portion of which should be rubbed in gently over the surface of the enlarged joints, and in some cases it is followed by diminution of swelling. I have used it, in addition, in an attempt to reduce the unsightly nodosities on the terminal phalanges, but the efficacy of this has been more apparent to my patients than to myself. Bruce recommends the following treatment for Heberden's nodes :—The affected fingers should be wrapped in boracic lint, previously soaked in a saturated solution of sodium chloride, then covered with oiled silk and bandaged. The bandage should be left on all night, and the method must be used for a considerable time if good results are to be obtained.

MASSAGE.

Massage is of great value, especially in the localized forms of the disease. Its use should be chiefly directed towards improving the nutrition of the wasted muscles, and diminishing their stiffness on movement. When the disease is confined to the knees, the wasting is chiefly in the extensors, and the efforts of the rubber should be concentrated especially on this group. Often the thigh muscles in these cases show no apparent wasting, but by means of electrical testing it is often possible to demonstrate delayed reaction time, which appears especially early in the vastus internus. In these cases, therefore, it is well to combine local electrical treatment with the massage. When the hip is affected, similar treatment is called for, and in this connection it must not be forgotten that the buttock must be included in the massage, as marked wasting is often present in the glutei. Massage to the joints themselves is only permissible when the disease is not active and the joint displays no irritability, and it should always be performed with exceeding gentleness.

ORTHOPÆDIC AND SURGICAL.

A glance at the underlying pathological condition in this affection soon convinces us that if judicious movement is essential to the well-being of the rheumatoid joint, judicious rest is the salient factor in the treatment of osteo-arthritis.

In the former we are faced with an ever-present tendency to fibrous and bony ankylosis with contracture if rest be unduly favoured and motion neglected.

On the other hand, in the latter we are menaced by the never remote contingency of fresh bony proliferations, villous outgrowths, and recurring synovial effusions, if we omit fixation wholly from our treatment and advocate constant movement of the affected articulation.

In the acute stages, when the joints are painful and irritable, exercise must be forbidden, and means taken to ensure a more or less complete degree of fixation of the articulations. Some authorities, like Goldthwait, consider that this should be absolute for a time, if joint symptoms are very decided. As a rule, protection of the joint, or limitation of its movement, is sufficient. Naturally, it is in the joints of the lower extremities that such is usually called for. In irritable states of the knee-joint, while complete rest is indicated for a time, a well-fitting knee-cap should afterwards be worn throughout the daytime when the patient has risen from bed. In milder cases, firm strapping or bandaging is all that these patients will put up with, and though not so effectual as a well-fitting knee-cap, it undoubtedly protects the joint, and diminishes the sense of insecurity that they complain of.

The plaster and leather retentive apparatus advocated by Locke and Osgood is only indicated if an unusually sensitive condition of the joint prevails.

Hoffa[4] recommends that some form of ensheathing apparatus should be worn continuously for a year. It should be discarded only during the night, and re-applied in the morning after massage, hot-air treatment, and gymnastic movements have been practised.

Naturally, this method of fixation achieves the best results when treatment is instituted early. It can, of course, be only expected to check and not to cure the underlying tendency to morbid proliferation of the cartilaginous and bony structures.

When patients refuse to adopt any such means of support, they should be warned of the lamentable consequences that may attend sudden strains of the affected joints.

Excessive synovial effusion is rare, and aspiration, though it has been practised, is seldom called for. Where such occurs frequently with symptoms suggestive of locking, the possibility of villous outgrowths being present must be considered.

Buedinger[5] has practised the injection of sterilized vaseline, in quantities of 1 to 4 cc., into the knee joint, and, in smaller amounts, into other articulations. He claims that, after a slight initial reaction, increased freedom of movement results.

Arthrotomy for the removal of villi is but rarely called for in this disease as compared with rheumatoid arthritis.

On the other hand, operation for the removal of bony spurs is sometimes indicated, either because of their actual intrusion between the bony surfaces causing pain and recurring effusions from internal traumatism, or when, as sometimes occurs, they have become free in the joint cavity and act as foreign bodies.

Osteophytic outgrowths may also be present external to the joint cavity, and may give rise to pressure neuralgia, or even definite neuritis. Tubby[6] relates two examples in which such outgrowths, situated on the posterior surface of the acetabulum, caused intense sciatic pain. He strongly advocates their removal where possible. Their presence was established by X-ray examination. Goldthwait also records examples of their interfering with the normal function of the joint, and advocates their removal.

Excision of the hip in inveterate cases of malum coxæ senilis has been performed by Riedel, Küster, W. Müller, and others. Opinions, however, differ as to the efficacy of the procedure. Hoffa, who has performed the operation in a series of cases, considers the results to be excellent, the patients being enabled to walk, having voluntarily abandoned their crutches. According to Tubby's experience the results are poor.

TREATMENT BY BATHS.

This is often attended by considerable benefit in this malady, whether it be of the generalized or localized variety. Even in the acute stages, when localized, hot douching of the affected joint is extremely soothing and beneficial, provided that no strain is placed upon the articulation, the patient being carried to and from the bath. When the disease is acute and many joints are involved, treatment by baths is not possible until the more severe symptoms have subsided.

Following this, however, total immersion in hot mineral baths, combined with undercurrent douches at a higher temperature, impinging directly upon the more painful joints, often avails much.

In the more chronic stages more vigorous methods may be adopted, such as a course of Aix-massage baths followed by a gradually cooled needle douche, a mode of treatment which I have often found of conspicuous benefit. Berthollet, or natural vapour baths, are also extremely serviceable, especially in aged persons who are too feeble to stand the higher temperatures employed in the hot-air and radiant-heat treatment. These admit of either generalized or localized exposure to the hot vapour. It is in osteo-arthritis, whether localized or generalized, rather than in rheumatoid arthritis, that good results are to be obtained from hot-air or radiant-heat baths. When only one joint is giving trouble a local bath is sufficient; but when many articulations are implicated, the whole body must be exposed to the superheated air.

Finally, by these methods of balneotherapy, usually not easy of access save at a well-equipped spa, much may be done to retard or arrest the progress of the disease, if not too advanced. It is, however, difficult, if not impossible, to foretell, even in severe cases of the affection, whether some degree of palliation may not be obtained by balneotherapy. I have even in long-standing cases of hip trouble frequently heard the sufferers attest that their feeling of painful infirmity was distinctly ameliorated, and this being so, I think that where such a method of treatment has not been adopted it should, in the absence of any contra-indications, be given a trial. Among other spas of Great Britain, Bath, by reason of its comparatively equable temperature and the natural heat of its waters, is especially suitable for this class of patients, who not uncommonly are somewhat stricken in years. Moreover, when, either through infirmity or financial circumstances, such sufferers are debarred from spending their winters abroad in warmer and drier climates, its undoubted and well-established claims for consideration should not be overlooked.

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SECTION IV.

SPONDYLITIS DEFORMANS.

SECTION IV.—SPONDYLITIS DEFORMANS.

(Syn. Vertebral form of Arthritis Deformans; Chronic Spinal Rheumatism, etc.)

CHAPTER XVII.

INTRODUCTORY AND HISTORICAL.

THE following section will be devoted to a consideration of the clinical and pathological aspects of the vertebral types of arthritis deformans.

Although for the convenience of description this central or spinal form is treated of separately, it must be clearly understood that no sharp line can be drawn between it and the peripheral types of arthritis deformans, owing to the existence of endless intermediate or transitional forms. All that can be alleged in distinction is that in the vertebral variety the brunt of the morbid process falls upon the spinal column, the disease in some instances being strictly confined to it, but more commonly implicating at the same time to a variable degree the joints of the limbs.

Under the title of spondylitis deformans there have doubtless been included many examples of chronic spinal rigidity which, strictly speaking, are not admissible into this category, as for example the various forms of infectious spondylitis-typhoid spine, etc.

The exact relationship of infectious spondylitis to spondylitis deformans has yet to be defined, but the clinical content of the former group has of late years increased so rapidly that undoubtedly a large number of cases, hitherto grouped under the latter, will have to be relegated to the former class.

While upon this question of the differentiation of the mass of diseases included under the former title, one must allude to the important researches of Strumpell, Marie, and von Bechterew in this connection.

Into a detailed description of the types of chronic spinal rigidity isolated by these observers, one does not propose here to enter, but would simply note the fact that both Strumpell and Marie, as well as von Bechterew, maintain that their varieties are separate entities, distinct not only from each other, but also from the vertebral type of arthritis deformans.

Osler, on the other hand, insists that neither the von Bechterew nor the Strumpell-Marie type are to be regarded as distinct diseases, maintaining that they are both types of arthritis deformans.

At first sight, it would appear as if these different views were so directly opposed as to admit of no compromise, but a brief digression will, I hope, tend to show that they are not wholly irreconcilable. Thus Marie and Léri claim to have established a pathological differentiation between the Strumpell-Marie and von Bechterew types on the one hand and "chronic spinal rheumatism" on the other.

From their researches it appears clear that in the two former varieties the bony changes in the bodies of the vertebræ are predominently atrophic, whereas in the latter they are hypertrophic in character.

The descriptions as well as the plates given by these authors of the latter or hypertrophic form seem to indicate very clearly that the condition designated "chronic spinal rheumatism" was one of osteo-arthritis of the vertebral column.

I am strengthened in this opinion by the fact that Teissier suggests for it the term "vertebral osteophytic rheumatism," as denoting the salient pathological characteristic of the affection.

Now, while few will dispute that Marie and Léri are justified in drawing a distinction on these grounds between the above types and osteo-arthritis of the vertebral column, to my mind it is not established that the types under discussion are to be regarded as distinct from the chronic spinal rigidity which arises in the course of a rheumatoid or atrophic arthritis.

From my own clinical observations, I am inclined to believe that the Strumpell-Marie type (la spondylose rhizomélique) and the von Bechterew variety are closely allied to, if not identical with, the rheumatoid or atrophic variety of spondylitis.

From the above considerations it would appear that there are two main divisions of spondylitis deformans, one rheumatoid or atrophic and the other hypertrophic or osteo-arthritic spondylitis. Under the former may be classed the Strumpell-Marie type, the von Bechterew, and those types of chronic spinal rigidity arising as part of a generalized rheumatoid arthritis, while to the latter will be relegated all cases of osteo-arthritis of the vertebral column.

This much-debated point of the further differentiation of spondylitis deformans will be treated more in detail in the succeeding chapters, and we now pass on to consider our subject from its historical aspect.

That this peculiar affection of the spinal column is one of great antiquity has been shown by Prof. Elliot Smith,[1] Bland-Sutton,[2] and others. To the extensive researches of the first-named observer in Nubia we have already referred in the opening section of this work when we noted the striking frequency with which evidences of arthritis deformans were found by him in skeletons exhumed during the process of exploration.

To this must be added the further and still more interesting observation by the same authority that the particular variety of the disease most commonly in evidence amongst these archaic remains was spondylitis deformans. The extreme prevalence of this vertebral type is shown by the fact that in one of the prehistoric cemeteries explored, every adult body presented signs more or less extensive of its effects. While the whole spinal column was in some instances affected, the site of election appeared to be in the lower lumbar vertebræ, though instances were revealed in which the morbid process was localized to the cervical or dorsal segments.

At the present day, according to Prof. Smith, the disease is commonly to be observed in Nubian people (*Plate XXVII*), especially amongst dwellers on the banks of the Nile, in whose waters the inhabitants have from time immemorial been wont to immerse themselves for prolonged periods in the process of their ablutions. To this traditional custom he believes may be referred the peculiarly marked incidence of the affection among these races.

Bland-Sutton also has described identical lesions in the spinal column of a mummy taken from one of the pyramids.

Again Virchow,[3] while conducting the antiquarian researches at Marienthon before referred to, brought to light a skeleton showing similar evidences, and in this case the bony outgrowths projected so markedly into the spinal canal as to leave that great pathologist in no doubt that the subject must during his lifetime have shown signs of paraplegia.

Regnault,[4] during the course of some research prosecuted at the Ecole d'Anthropologie, noted that out of a series of sixty-three skeletons of different nationalities, seventeen presented the anatomical characteristics of spondylitis deformans. Among the races represented were Chinese, Arabs, Hindoos, and others, thus showing that the disease occurs indiscriminately, no individual race apparently enjoying immunity. Its incidence in members of the animal kingdom has been noted by several authorities. Thus Struthers[5] observes its ravages in the vertebræ of whales, while Sutton[6] reports its occurrence in oxen and horses, and Bricon[7] describes a similar condition in cats.

The earliest detailed study of the morbid anatomy of the affection that I have come across is one recorded in the *Philosophical Transactions* (1700) by Dr. Bernard Connor,[8] under the title, " The Bones of a Sceleton united without Joynting or Cartilage " (*Plate XXVIII*). His account begins as follows :—

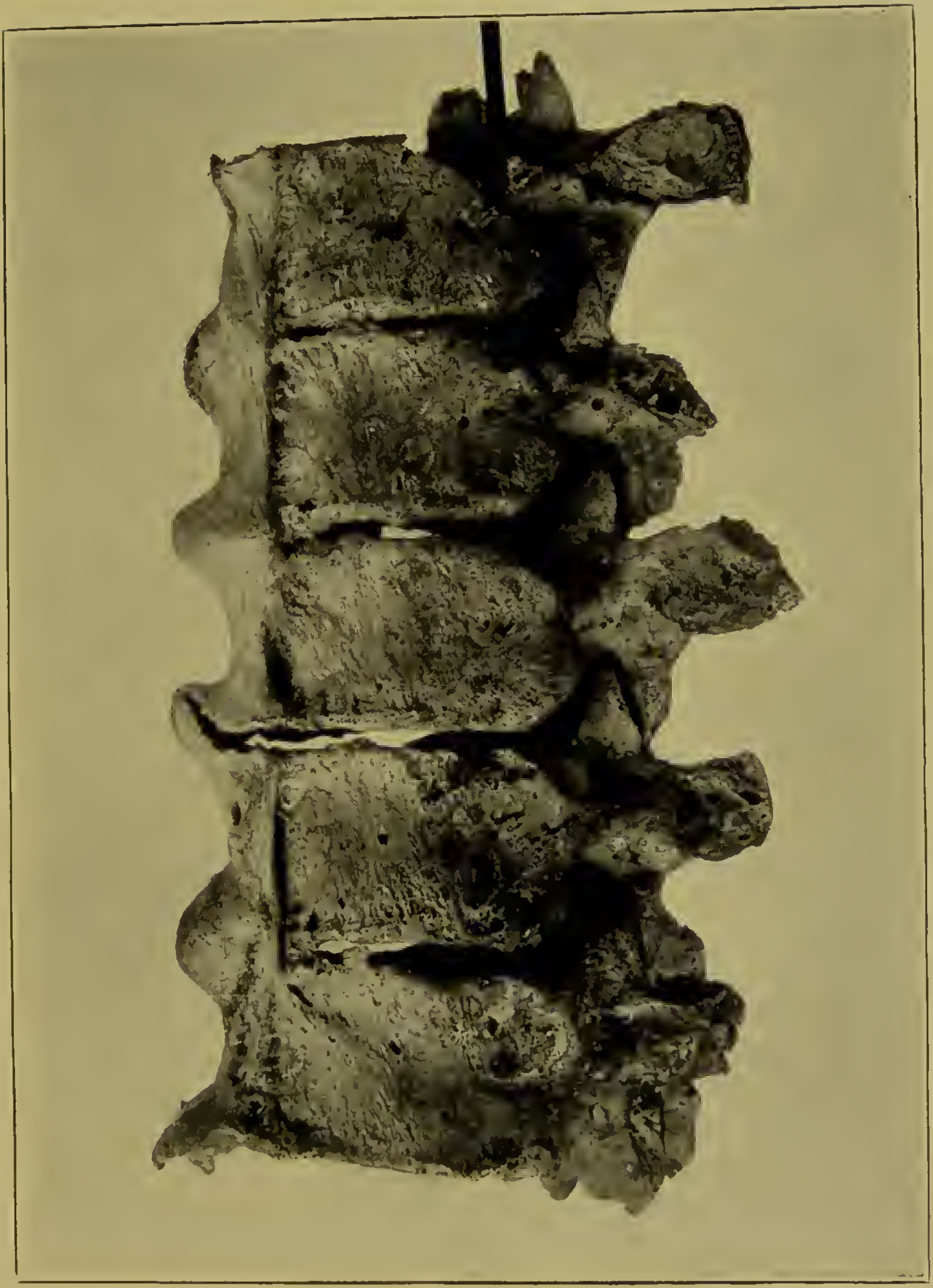
" I have lately seen in France part of an human sceleton, consisting of the os ilium, the os sacrum, the five vertebræ of the loyns, ten of the back, five entire ribs on the right side and three on the left ; the bottom or ends of the other were closely united to the transverse apophyses of their vertebræ ; the vertebræ of the neck, the clavicle and sternum were wanting."

In his description of the spinal column he states : " All these bones which naturally are separate and distinct from one another were here so straightly and intimately joyned, their ligaments perfectly bony and their articulations so effaced that they really made but one uniform continuous bone, so that it was as easie to break one of the vertebræ in two as to disjoynt or separate it from the other vertebræ, or the os sacrum from those of the ilia.

" The roots of all the ribs made but one equal smooth and plain superficies with the vertebræ and their apophyses.

" The oblique apophyses of the vertebræ were so confounded

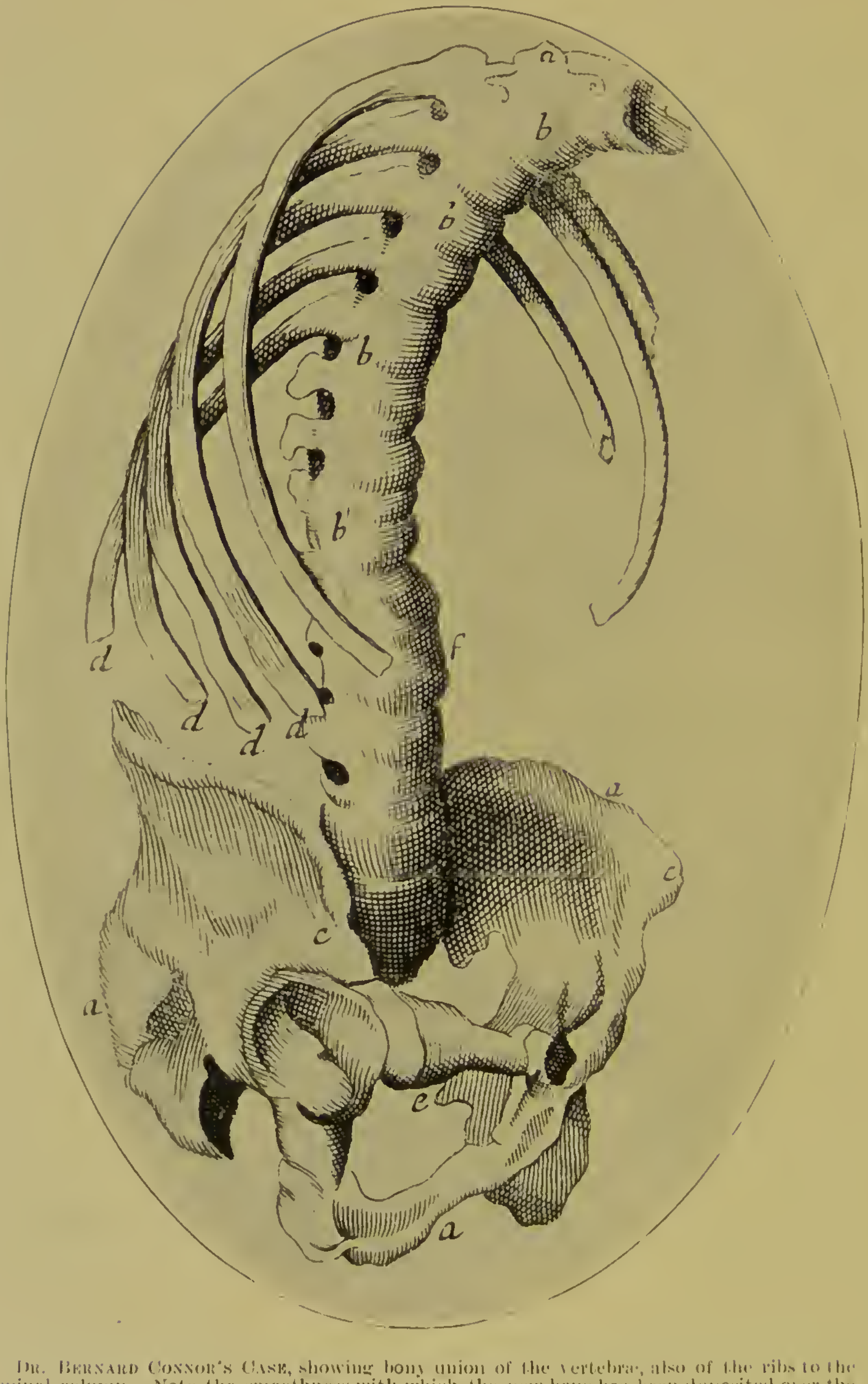
PLATE XXVII.



Specimen of Lumbar Portion of Spine, from the Nubian collection of Prof. Elliot Smith, showing bony union of the vertebræ along their anterior surfaces.

(From R.C.S. Museum).

PLATE XXVIII.



Dr. Bernard Connor's Case, showing bony union of the vertebræ, also of the ribs to the spinal column. Note the smoothness with which the new bone has been deposited over the intervertebral and costovertebral articulations, the absence of osteophytes, also the sinking of the spine into the pelvis, and the ankylosis of the ribs to the vertebræ.

and lost that it was not possible to observe any marks of them.

"The cartilaginous edge of the vertebræ themselves were turned to perfect bone. But when I had saw'd two of the vertebræ asunder at the commissure I found that this uniting did not enter above two lines deep, and that afterwards their middles were separated as they usually are, and touched each other only at their edges, which was raised up a little above the middle part. The figure of this trunk was crooked and made part of a circle, the spine making the convex, and the inside of the vertebræ the concave part of this segment. The cavity for the spinal marrow had no fault but its bending figure."

The author also adds that there was no enlargement or "unnatural bunching out" of the vertebræ, "their joyning together very regular, no one vertebra standing out beyond the other, either before, behind, or on the sides." It would appear, therefore, that there were little or no osteophytic outgrowths in his case, and that the ankylosis was largely due to ossification of the ligamentous or capsular structures, thus approximating to that type of spondylitis known as spondylose rhizomélique, and which Leri, to adopt his own nomenclature, considers to correspond to "une menisco ligamentite."

It is also worthy of note that the old physician, from the data at his disposal, deduced some of the outstanding clinical features associated with the malady. Thus he says: "From this construction of the parts, the body of this person must have been immovable, that he could neither bend nor stretch himself out, rise up, nor lie down, nor turn upon his side." He also remarks that owing to the immovable ribs his breathing must have been short.

As regards its pathogeny the author inclined to think the deformity was due to malposition in utero.

Approaching the subject from the clinical side we also find that this marked tendency on the part of the disease to involve the vertebræ has long been recognized. Thus Haygarth,[9] in his treatise, notes that "in few patients a crackling noise was perceived in the joints when in motion, particularly in the neck."

Todd,[10] in his work, also cites an instance in a girl, æt. 25, in whom all the joints, including the cervical vertebræ, were attacked, and the resulting crippledom was so deplorable that machinery had to be constructed to enable her to be raised from bed.

Neither of these observers, however, seems to have met with cases in which the manifestations of the disease were absolutely limited to the spinal column. Adams,[11] on the other hand, evidently recognized this variety, and, when considering the anatomical characters of the affection, points out that those portions of the spinal column which, in a normal state, enjoy a comparatively greater play of mobility, are precisely those in which traces of the disease are most commonly met with—hence the cervical and lumbar articulations are more frequently involved than the dorsal. Of those of the cervical region, moreover, he observes that by reason of their relatively still more constant motion they display evidences of this disease on their articular surfaces more often than all the other segments of the column taken collectively. Nevertheless this observer made no attempt to differentiate this central or spinal type from those in which the joints of the column were only involved as part of a generalized arthritis, his studies of their morbid anatomy evidently having convinced him that the articular lesions in both cases were identical in nature, though differing in their mode of distribution.

It is, however, remarkable that in none of his cases does he seem to have noted the occurrence of the severe radiating pains so frequently to be met with in this vertebral type, and that he himself was somewhat surprised at the absence of such pressure symptoms is obvious in his writings. Thus when discussing one of his museum specimens, in which the spinal canal, and the foramen for the vertebral artery at the level of the first cervical vertebra, were markedly encroached upon by bony enlargement of one of the oblique processes, he states: "It is not known that any symptoms which might be supposed referable to such a state of things have ever been observed."

Just about this time (1857–58) Sir Samuel Wilks[12] drew attention in the *Guy's Hospital Reports* to the frequency with which in labouring men the anterior surface of the spine is covered by "a liquid osseous substance," and made the interesting observation that in such cases the articular processes were but seldom the seat of bony outgrowths.

With regard to the sensory accompaniments of the malady, it would appear from Oppenheim's[13] writings on the subject that some at any rate of the older authors were quite alive to their occurrence. This author in his work on *Diseases of the Nervous System* devotes a small section to arthritis deformans of the vertebral column, in which special emphasis is laid upon the severity of the nervous symptoms, and at its conclusion he states that all the clinical facts described by him had been fully considered by Braun[14] in 1875.

Historically speaking, this statement of Oppenheim in regard to Braun's work is of interest, because of the similarity obtaining between Oppenheim's group of cases and those described by von Bechterew, of which we have heard so much recently, inasmuch as it tends to support the contention of some, that from a clinical point of view the type now associated with the name of Bechterew had long been recognized.

Individual examples of the affection were reported by Davies Colley[15] (1855), Hilton Fagge[16] (1877), and Sturge[17] (1879). In Fagge's patient, who died of a chest affection, the autopsy revealed complete ankylosis of the arches and the spinous and transverse processes of the dorsal vertebræ, likewise ankylosis of the costovertebral attachments and the right hip joint. Great softening and fragility of the bones of the column were also a feature. Special interest attaches to this example in that the hip joint was involved as well as the column, the case therefore approximating to that variety of spondylitis known as spondylose rhizomélique.

An additional resemblance is afforded by the marked softening of the bones, a condition which, according to Leri,[18] is especially characteristic of this type of spinal rigidity.

Bradford,[19] in 1883, recorded three instances of "rheumatism of the spine," in all of which the disease was confined to the vertebral column, stating also that in two of the cases there was a history of gonorrhœa.

This authority and Broadhurst[20] appear to have been among the first to recognize the possible etiological relationship of gonorrhœa to this form of spinal ankylosis. One of the first persons to apply the term spondylitis deformans to the affection was apparently von Thaden,[21] who wrote on the malady under this title in the *Archiv. f. klin. Chir.*, iv., p. 565.

In 1884 Strumpell,[22] in the chapter on chronic articular rheumatism and arthritis deformans in his text-book, describes the type now associated with his name and Marie's, and for which the latter has suggested the term spondylose rhizomélique. Thus he writes: "A remarkable, and as it seems to us, unique disorder, may be mentioned in passing. It leads very gradually and painlessly to a complete ankylosis of the entire spinal column and the hip joints, so that head, trunk, and thighs are firmly united and completely stiffened, while all the other joints retain their normal mobility. It need scarcely be said that this necessarily causes a peculiar modification of the carriage and gait of the sufferer. We have seen two cases of this affection which resembled each other very closely."

Subsequently to Strumpell's work little of interest was forthcoming until 1892, when von Bechterew[23] published a paper entitled, "Rigidity and curvature of the vertebral column as a special form of disease," in which he endeavoured, like Strumpell, to effect a further cleavage of the various forms of spinal rigidity grouped under the terms arthritis deformans, articular rheumatism, and so forth. Inasmuch as I shall allude more in detail to this variety in the clinical section, it will be sufficient here to state that the salient characteristics of this type consist in a chronic rigidity of the spine, often limited to the cervical region, with, in addition, marked root symptoms, the other joints of the body remaining intact. The important part played by Strumpell and von Bechterew in directing attention to, and reviving interest in, the study of chronic vertebral diseases of this class, can hardly be over-estimated, and is abundantly proved by the increasing number of publications forthcoming on this subject since their researches were made known.

Among the more valuable contributions are those by Marie and Astie,[24] Donnetti,[25] Zenner,[26] Sachs and Fraenkel,[27] Koehler,[28] Leri, Oppenheim, Goldthwait,[29] and Gowers.[30] Marie and Astie, in 1898, reported a series of five cases under the title "spondylose rhizomélique," using the term to denote the frequency with which the shoulder and hip joints (root joints) were involved as well as the column. The name, however, is scarcely applicable, as since then examples have been recorded in which other articulations have been implicated. Owing to their identity with those previously described by Strumpell, this variety is often referred to under the name of the Strumpell-Marie type of spondylitis. These observers hold that their group is essentially distinct both from arthritis deformans and the von Bechterew type.

Oppenheim, however, maintains that the distinctions drawn are untenable, and while he admits that possibly in the future characteristic diseases may be found affecting the vertebral column without implicating any other joints, still for the present he is disposed to agree with Senator[31] that "all conditions described so far can be included under chronic articular rheumatism, gout, and arthritis deformans."

With this view many writers are in accord, among them Sachs and Fraenkel, who in their excellent résumé of the subject state that the changes found in "progressive ankylotic rigidity of the spine differ in degree and in localization, but not in kind, from those found in extreme forms of articular rheumatism and arthritis deformans."

Koehler reported a case in which hips, knees, and spine were absolutely rigid. He was able by palpation of the pharynx to detect exostoses on the sides of the cervical vertebræ during life, and their presence, together with the general appearance of the joints, led him to attribute the condition to arthritis deformans.

Zenner considers that some cases in his series were primarily arthritic, as evidenced by the presence of disease in joints other than those of the vertebral column, and, in such, a condition of gout, arthritis deformans, or rheumatism may be responsible. In others, however, it was not so clear as to where the primary lesion was situated, and in two of his cases he was disposed to view the condition as one primarily affecting the muscles, bony ankylosis ensuing secondarily to inflammation in the vertebræ or intervertebral discs, or, may be, inducing bony ankylosis in a like manner to that observed by Marsh[32] as occurring in lateral curvature. Beer,[33] quoted by Mutterer[34] and Zenner, was also inclined to consider the soft parts as the chief seat of the disease. Goldthwait records several examples of spondylitis deformans, and considers the condition as one of "osteo-arthritis of the spine," maintaining that some of the cases reported as such were undoubtedly instances of rheumatoid arthritis of these articulations, and not osteo-arthritis, to which he would confine the application of the term.

Leri, in the *Revue de Médecine*, contributes an extensive résumé on the subject of spondylose rhizomélique, and also an account of the post-mortem findings in one of Marie's cases, the main features of which were as follows:—Very marked curvature of the spinal column in the dorsal and cervical region, this portion being practically at right angles to the lower lumbar vertebræ; there was also some abnormal anterior curvature of the lumbar column; the ligaments were strikingly ossified, almost exclusively on the convexities of the curvatures, the lumbar lordosis showing ossification of the anterior portions of the disc, the dorsal kyphosis similar transformation of the supraspinous ligaments. In addition, there were marked hypertrophy and ankylosis of the articular extremities.

Now while most observers agree that the Strumpell-Marie type should be included under the heading of spondylitis deformans, not a few dispute the propriety of placing von Bechterew's group in the same category. Both Dana[35] and Hendrie Lloyd,[36] for example, are of opinion that the vertebral column is affected secondarily in these cases—in other words, that the curvature is of neuropathic origin, resembling those occurring as a result of various cord diseases, such as syringomyelia.

Sir William Gowers draws attention to the fact that narrowing of the foramina, with resulting compression of nerve roots, may occur, also that the cervical sympathetic may be involved, as evidenced by pupillary changes, but that the cord itself is seldom implicated.

Kummel[37] has described cases of spondylitis slowly developing after vertebral injury giving rise to angular deformity and radiating pains, and, may be, signs of compression. He considered them to be types of traumatic spondylomalacia; similar cases have been described by Henle,[38] Heidenhain, Schultze, and others. I have called attention to this group of cases inasmuch as Marie has attempted to identify these, as well as those of von Bechterew's type, with the condition designated by him "la cyphose hérédo-traumatique." Forestier,[39] as a result of his vast experience at Aix-le-Bains, in addition to the Strumpell-Marie and von Bechterew types, recognizes one of rheumatic nature, and one associated with arthritis deformans. He arranges the forms he has experience of under three headings: (1) Ankylosis; (2) Pseudo-neuralgic; (3) Simple.

Under the first heading he classes those where the onset is slow, gradual, and insidious, and in which pain, though an early symptom, soon disappears. Joints other than those of the spine are rarely affected. Exostoses are sometimes palpable, and the condition culminates in one of bony union with ossification of ligaments.

To the pseudo-neuralgic form I shall allude later, in the clinical account.

In the third form the rigidity, instead of being generalized, is localized to some portion of the spine, and he places in this category the more chronic forms of lumbago, and some types of torticollis. Pain is slight, rigidity not absolute, and the prognosis good.

In conclusion, the preceding historical résumé, though far from complete, does, I venture to hope, include the more important contributions on the subject; but for the convenience of those who would desire to study the original contributions more in detail I have appended a fairly ample bibliography.

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CHAPTER XVIII.

ETIOLOGY.

IN the majority of recorded examples the subjects of the affection have been of the male sex, only isolated instances of its occurrence in women and children having been noted hitherto.

As regards its causation it must be admitted that we possess little or no definite knowledge on this important point, and such facts as are at our disposal do not seem to indicate that there is anything distinctive about the etiology of spondylitis as opposed to all other forms of arthritis deformans. Prolonged residence amidst unhygienic surroundings, exposure to damp and cold, etiological factors both of them common to all other varieties of this disease, figure largely also in the life histories of sufferers from spondylitis, and in the opinion of some, constitute an adequate reason for the development of the malady. In one of my own cases a severe drenching seemed responsible for the onset of the affection, and Goldthwait comments on the frequency with which "osteo-arthritis" of the spine develops in persons whose occupation entails exposure to sudden extremes of temperature, such as stokers and the like. Many writers, however, are not disposed to consider these environmental circumstances as sufficient in themselves to initiate the disease, maintaining that an additional element in the form of an infection is essential, and that the former are to be regarded as predisposing rather than actual causes, which, by lowering the vis resistantiæ of the individual, pave the way for the occurrence of an infection.

According to Beucke,[1] senility and mechanical causes play an important part in its production, but obviously this explanation, because of the comparative youthfulness of many of the subjects, is capable of only a very limited application, and could only be entertained in regard to cases of senile kyphosis, which, as Garrod[2] suggests, probably form a special class apart from those under consideration. In the opinion of Sir Samuel Wilks,[3] the mechanical factor contributed greatly to the development of what he termed labourer's spine, and more recently Baumler,[4] discussing the etiology of a case of spondylitis under his own observation, enunciated the same belief. He points out that localized pain and tenderness of a more or less persistent character may ensue even in normal healthy spines when a faulty or strained attitude is maintained for prolonged periods.

In continuation he draws attention to the fact that in some cases of spondylitis, inflammation of the hips or a portion of the spinal column is present, and suggests that such a condition, reinforced possibly by the strain imposed upon the spine by some special occupation, may induce changes in the hitherto uninvolved or healthy spine, the lessened mobility leading to localized circulatory disturbances favouring congestion and subsequently inflammation consequent on infection by micro-organisms.

The influence of heredity and trauma have been enlisted to explain the development of all varieties of spondylitis deformans, but an attempt has been made to specially identify these particular etiological factors with the type described by von Bechterew.[5] In his group of cases there were certainly some indications of a possible hereditary or familial tendency; thus in one of his subjects, a woman fifty years of age, the patient's mother and son were both similarly afflicted; in another instance mother and son showed the same spinal affection. Marie and Astie[6] also insist upon the important rôle played by heredity and trauma in the etiology of this variety; indeed, so convinced are they of the validity of von Bechterew's claim that they designate this type by the term "la cyphose hérédo-traumatique." These authorities have recorded an instance in which three members of one family were affected, namely, father, son, and daughter. Warrington[7] has recently reported two examples of "chronic rigidity of the spine" the subjects of which were brother and sister, while Seguin[8] observed its incidence in three children of the same family.

In this connection it is interesting to note that Sir William Bennett[9] considers that "congenital stiffness of the spine" is by no means as rare as is generally supposed. The rigidity in such cases, so he states, is almost invariably limited to the dorsal or dorso-lumbar region, and, moreover, displays a tendency to affect more than one member of the same family.

Kirchgaesser,[10] discussing the etiology of a series of cases, the clinical characters of which closely resembled those of the spondylose rhizomélique type, suggests the possibility of a congenital predisposition. Inasmuch as many of the foregoing cases were not strictly speaking of the von Bechterew type, it cannot be claimed that this hereditary or familial mode of distribution is peculiar to this particular variety of spondylitis. In my own experience I have not observed any such indications, and from a review of the recorded examples of spondylitis deformans am led to the conclusion that hereditary transmission of this disease is extremely rare, and that such influences therefore cannot be accorded a prominent share in its production.

Passing now to consider the other prime etiological factor, "trauma." In von Bechterew's series there were certainly indications of such a causal relationship, the disease in some instances supervening somewhat quickly after an injury to the back.

Both Marie and Astie also lay stress on the frequency with which this sequence occurs, alleging in further support of their contention that the clinical course pursued by cases of this group closely resembles that observed in "traumatic" affections of the vertebral column.

It must, however, be remembered that these authorities, as shown in our historical section, place a very wide interpretation upon their term "la cyphose hérédo-traumatique," for not only do they include von Bechterew's group under this designation, but also a number of other cases which most authorities would class as examples of "traumatic kyphosis," traumatic spondylomalacia, etc. In considering, therefore, the relationship of trauma to this type of spondylitis, Garrod's warning that gross injuries to the spine may give rise to a condition which closely simulates that resulting from spondylitis deformans must be borne in mind.

It is convenient here to refer to the different views held as to the pathogeny of this form of spondylitis.

Von Bechterew held that the disease began primarily as a spinal meningitis, with resulting compression of the nerve roots, followed by secondary cord changes, atrophy of intervertebral discs, and ultimately ankylosis.

Marie and Astie likewise suggest that the condition is of neurotrophic origin, the nutrition of the vertebræ becoming impaired secondarily to concussion of the spinal cord following traumatism. Another hypothesis, originated by Mickulicz[11] and Henle,[12] is that in a certain number of cases of this description it is possible that as a result of trauma an extra- or intra-dural hæmatoma is produced which, by the pressure exerted upon the nerve roots and ganglia, initiates certain sensory and trophic disturbances that culminate in a localized softening of the vertebræ. The suggested modes of pathogeny put forward in the last two views seem to still further justify the suggestion that some, at any rate, of Marie and Astie's cases were examples of violent injury to the spinal column, and cannot therefore be legitimately included with von Bechterew's group, as examples of the vertebral type of arthritis deformans.

As to the relationship of trauma to spondylitis deformans considered as a whole, if we except the instances reported by von Bechterew, Marie, and Astie, the number of recorded examples ascribed to injury is so small as to preclude its being regarded as an important, much less an essential, factor in its genesis. In all probability, as Poncet[13] and Leriche, when discussing the etiology of the affection, suggest, the action of trauma is effectual only in so far as it favours infection.

It now remains for us to consider the etiological relationship of various infective disorders to spondylitis deformans. That there existed some obscure connection between these two groups of disease has long been suspected by many students, and it must be admitted that recent bacteriological work distinctly favours the view that some cases at any rate of this description are of infective origin.

The researches of Fraenkel[14] have taught us that infections other than tubercle and syphilis may attack the vertebræ, and it has been shown that the spinal marrow may be invaded by a variety of organisms—pneumococci, typhoid bacilli, staphylococci, and others. Such have been identified *in situ* and cultures of them obtained from the medullary substance of the bodies of the vertebræ.

Labeyrie,[15] in a recent contribution to the *Gazette des Hôpitaux*, entitled, "Non-tubercular Osteitis of the Vertebral Column in

Adults," strongly urges the infective nature of spondylitis, including as one of his types "a chronic form of ankylosing osteo-arthritis," which he believes to be closely related to gonorrhœa and acute rheumatism. Broadly speaking, these cases of infectious spondylitis fall into two groups—suppurative and non-suppurative. This last type is characterized by pain and swelling over the affected vertebræ, usually the lumbar, with sensory and motor phenomena in the lower extremities. Now while the more acute and suppurative varieties of osteomyelitis of the vertebral column are hardly likely to be confounded with spondylitis deformans, it is not only possible, but highly probable, that some cases now called spondylitis deformans, especially those affecting only a portion of the spinal column, will eventually be classed as examples of non-suppurative infectious osteomyelitis of the vertebral column.

As we saw when dealing with the peripheral as opposed to this central or vertebral form of arthritis deformans, one of the great obstacles to further differentiation consisted in the impossibility of drawing an absolute distinction between the more acute forms of rheumatoid and osteo-arthritis and the so-called infective arthritides, and here again in spondylitis deformans we are confronted with the same problem.

A satisfactory solution on purely clinical grounds is hardly to be expected, but there is reason to believe that future bacteriological researches will establish a criterion. Thus McCrae[16] has already demonstrated the fact that spondylitis may follow typhoid fever, and by radiography has shown that such may be accompanied by definite bony changes in the vertebræ. This being so, it seems not unlikely that spondylitis will be found to ensue subsequently to other specific infectious disorders. The infections most commonly cited in connection with spondylitis deformans are those of gonorrhœa, rheumatism, syphilis, and tubercle.

As regards gonorrhœa, many writers have commented on the frequency with which it figures as an antecedent, but it would appear from a review of the literature that there is a tendency to exaggerate its importance, as in the majority of instances this assumed cause is absent. Marie and Leri, however, in a recent study of spondylose rhizomélique, state that the predominant infection in this type is one of gonorrhœa.

Both the acute and chronic forms of rheumatism have been assigned as causes, and certainly in my experience a history of previous arthritic affections is not uncommon. Leaving aside the question as to the exact nature of the preceding joint affection, it is certain that the history of an antecedent polyarthritis is forthcoming in not a few examples of this vertebral type of arthritis deformans.

In a paper on spondylitis deformans contributed to the *Edinburgh Medical Journal* I stated that cases of this description might begin as a polyarthritis, and either subsequently or coincidently the vertebral joints become involved. The further course of these cases is variable. In some with only slight spinal involvement the vertebral joints often recover mobility, and the disease lingers on in the joints of the limbs. In others the joints of the extremities recover, leaving the vertebral articulations permanently ankylosed, and this immobility may affect the whole or only a portion of the spinal column. In this respect my experience accords with that of McCrae, who also has observed that the subjects of spondylitis may have suffered previously from a polyarthritic affection. As a rule the polyarthritis of the limbs antedates by some considerable period the implication of the intervertebral joints, but in one of my cases the spinal rigidity supervened actually during the second attack of what was called " rheumatic fever." L. N. Müller[17] describes a similar instance in a young man in whom, after an attack of " acute inflammatory rheumatism," there gradually arose a rigid condition of the spine until the whole column was fixed.

Again in a series of fifty-eight cases of spondylitis deformans collected by Braun,[18] in the majority of whom the disease was confined to a portion of the spinal column, chronic rheumatism was assigned as a cause in no less than forty-six, and of the remaining, three were ascribed to syphilis, four to gout, and three to arthritis deformans.

G. Kirchgaesser reported a number of examples of " inflammatory necrosis of the spinal column," approximating very closely in their general characteristics to those of spondylose rhizomélique, stating that in his opinion the cause was probably often rheumatism, but that in some cases gonorrhœa appeared to have been concerned. Senator[19] and Oppenheim[20] agree in regarding the condition as due either to chronic articular rheumatism, gout, or arthritis deformans. The former also adds that the nerve symptoms in these cases may be due either to pressure on the nerve roots or to inflammation of the spinal cord of a gouty or rheumatic nature, and that these may occur quite independently of the disease of the vertebræ.

The foregoing facts show that "chronic rheumatism," so-called, is one of the most frequent precursors of spondylitis deformans. It must, however, be borne in mind that the term itself is extremely vague, and one that admits of wide differences in interpretation. For this reason it is, I think, much to be regretted that in so large a number of cases in which it is assigned as the cause, it is not specified whether the condition was one due to chronic articular mischief or one accompanying chronic muscular rheumatism. As Oppenheim points out, the latter condition may give rise to a condition of spinal rigidity, one moreover indistinguishable from "chronic articular rheumatism," unless resort be had to an anæsthetic, under the influence of which the rigidity, if due to muscular spasm, disappears, but, on the other hand, remains unaltered when the immobility is the result of articular mischief.

Personally I cannot say that I have ever met with a condition of permanent and extensive spinal rigidity which I felt justified in ascribing to a condition of "chronic articular rheumatism," as in all the cases of this description which I have seen, the immobility appeared to me to be the result either of rheumatoid arthritis or osteo-arthritis.

The etiological relationship of syphilis has chiefly arisen in regard to the type of von Bechterew, some authorities, Dana,[21] for example, disputing the propriety of its inclusion within the category of spondylitis deformans, inasmuch as the changes in the vertebræ were secondary to disease in the cord or its membranes, and suggesting that the morbid appearances revealed in the only case of von Bechterew's which came to autopsy were those of specific meningitis.

The possibility of a tubercular origin is chiefly advocated by Poncet, Leriche, and others of the French School, but hitherto this view has not obtained wide acceptance. In support of their contention they refer to a contribution by Lorentz, who considers that the frequency with which vertebral ankylosis occurs in tuberculous patients is quite inexplicable on the ground of mere coincidence, inasmuch as in a series of 174 phthisical patients he had found rigidity of the spinal column in no less that sixty-eight, of whom 55 per cent were under forty years of age. In my own experience I have only met two examples of spondylitis in which there was any evidence of tubercular mischief.

In reviewing this brief abstract of the etiology of spondylitis deformans one notes that while some authorities claim that environmental circumstances, mechanical causes, and traumatisms are in some obscure way causally related to this vertebral type of arthritis deformans, yet by the majority these factors are regarded effectual only in so far as they cause direct damage to the nervous system or favour the occurrence of an infection.

As we saw in the case of the von Bechterew type, some believe it to be of neurotrophic origin, while others attribute it to an infection, and a similar diversity of opinion obtains in regard to the pathogeny of the Strumpell-Marie group. There is, as before stated, nothing inherently antagonistic in these two views, and in my opinion the exigencies of the etiological problem would be better met by a blend of the two views ; in other words, that we should regard the underlying pathological condition as of the texture of an infectious or toxic trophoneurosis, the microbe or toxins acting either directly upon the cord or nerve roots, or indirectly through the medium of a spinal meningitis, to induce disturbances of nutrition in the vertebræ.

I am led to such a view of their pathogeny by certain clinical resemblances, in the aspect and symptoms of these cases, to those observed in extensive spinal rigidity arising in the subjects of rheumatoid or atrophic arthritis.

Moreover, judging from recent post-mortem observations made by Marie and Leri, there is not only a clinical, but also a pathological, similarity, in that both in the von Bechterew and the spondylose rhizomélique type, the essential bony changes in the bodies of the vertebræ are atrophic in character.

It may, however, be objected in regard to the foregoing hypothesis as to the pathogeny, that spinal meningitis is not a feature of rheumatoid arthritis, but in return I would refer to the work of Teissier,[22] Le Roque, Lepine, and others of the French School who maintain that it is by no means rare in this affection.

As to an etiology of osteo-arthritis of the spine as opposed to the foregoing atrophic varieties, we possess little or no information, and until our knowledge of the pathogeny of the more easily studied peripheral form of osteo-arthritis becomes more exact, it is hardly likely that any further light will be thrown on this very intricate problem.

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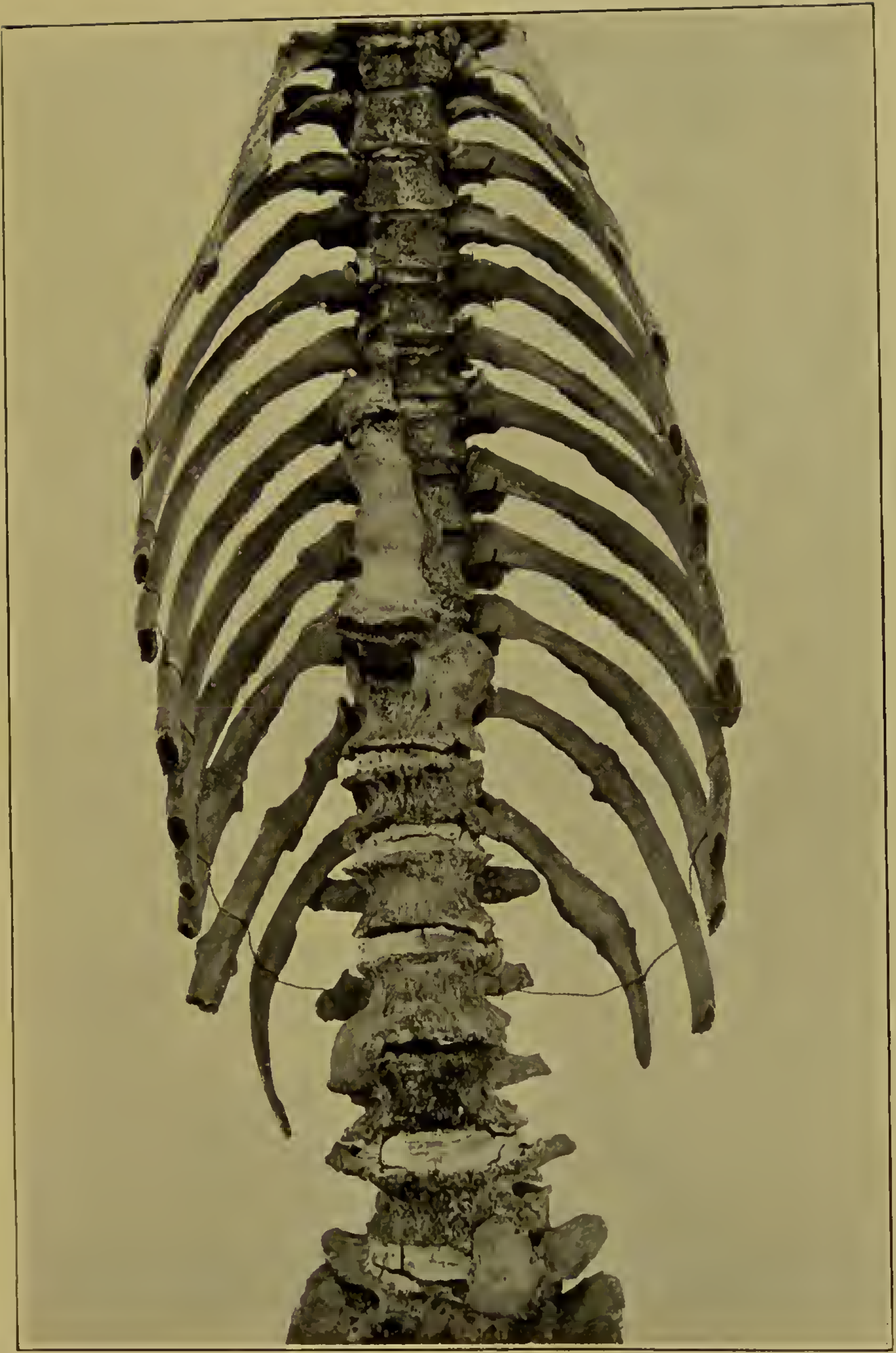
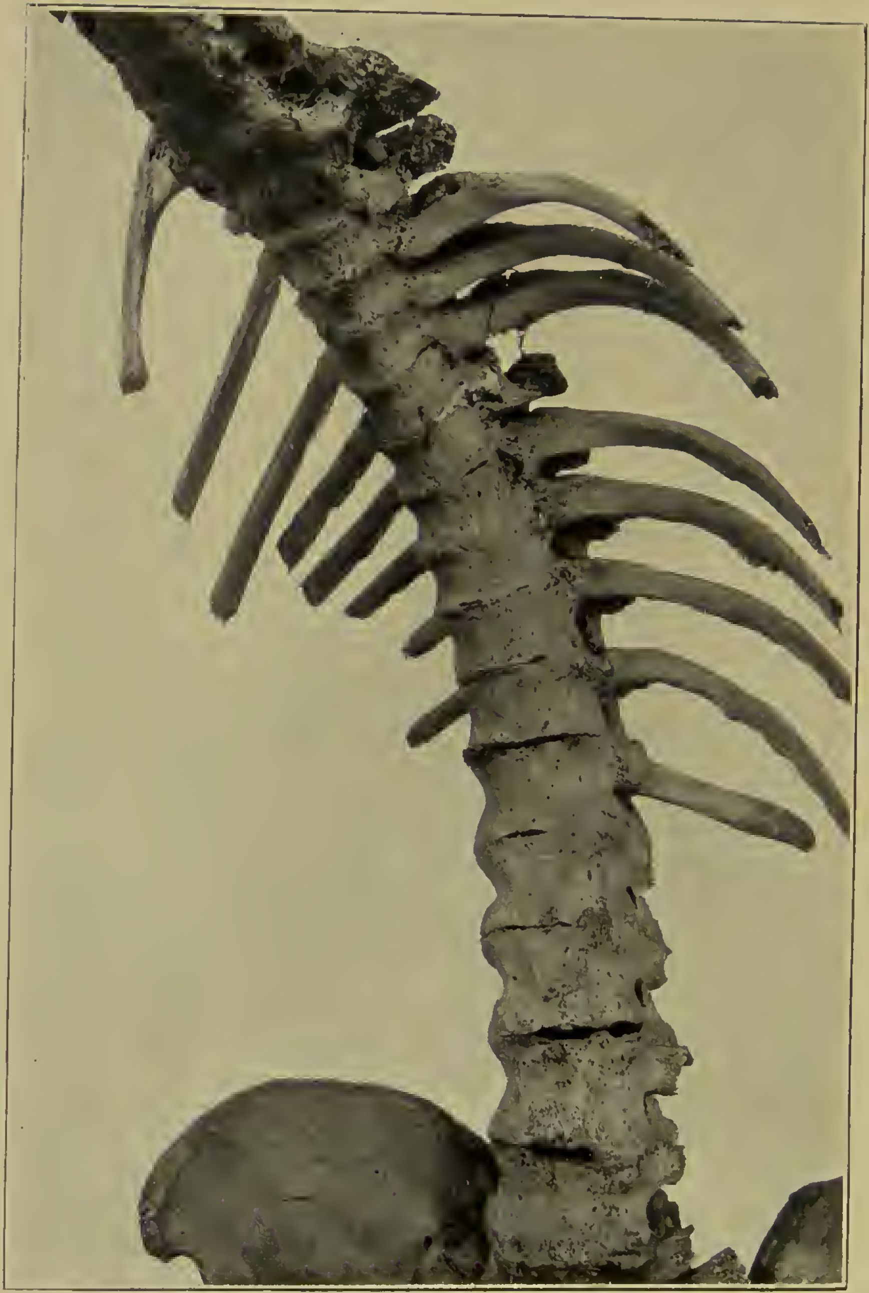
PLATE XXIX.



Vertebral Column with Part of the Ribs, showing plates of bone situated on the sides of the bodies of the vertebræ. The 7th, 8th, and 9th dorsal vertebræ are thus united by a smooth plate of bone, which is nearly continuous with a similar formation on the bodies of the 6th and 10th vertebræ. The last cervical and 1st dorsal, 10th and 11th dorsal, 2nd and 3rd lumbar vertebræ similarly united. No distortion of spine. Several old fractures of ribs present.

(From R.C.S. Museum. No. 2049).

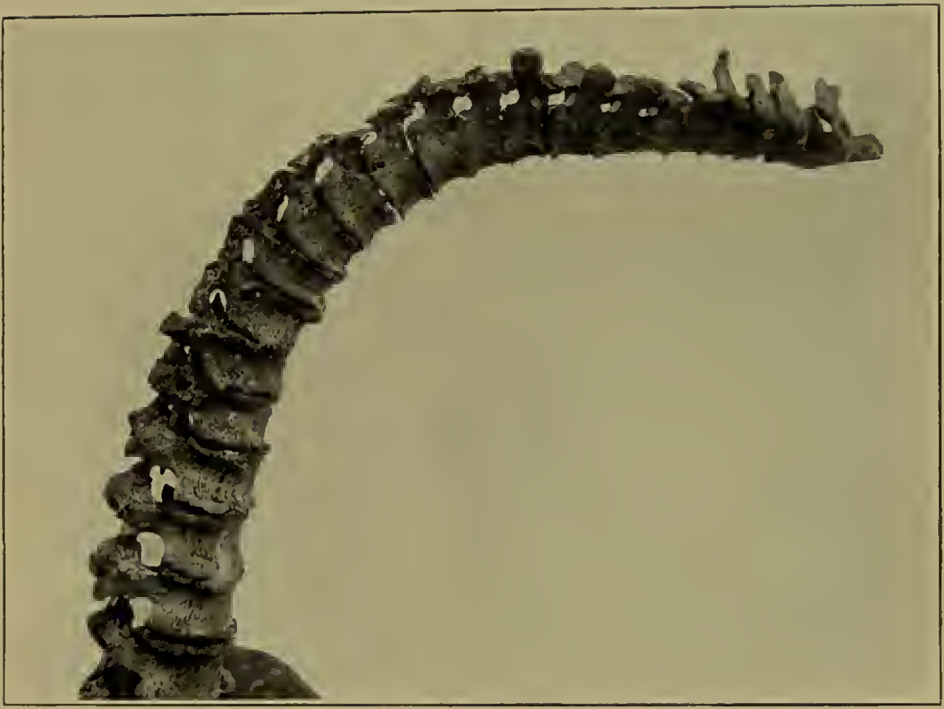
PLATE XXX.



Vertebral Column and Pelvis, showing coalescence of all the vertebræ and all the ribs with the spinal column by plates of bone extending over all their articulations. Sacro-iliac joints are also almost completely ossified. In only a few parts of the dorsal and lumbar regions are there present spaces in the position of the intervertebral ligaments, and only in the upper cervical and lower lumbar regions are there any apertures where the ligamenta subflava were. Intervertebral foramina are of natural size. Natural anterior curves of cervical and lumbar regions are obliterated, the spine being not otherwise deformed. Note smoothness of osseous deposit and absence of osteophytes.

(From R.C.S. Museum No. 2050).

PLATE XXXI



VERTEBRAL COLUMN AND UPPER PART OF PELVIS OF AGED WOMAN. With pelvis in the erect posture the second cervical vertebra is eight inches in front of the symphysis pubis, the column forming a single kyphotic curve. New bone formation has occurred on the bodies of nearly all the dorsal and lumbar vertebræ, in some cases leading to fusion of the contiguous segments. Intervertebral foramina not encroached upon.

(From R.C.S. Museum. No. 2107).

PLATE XXXII.

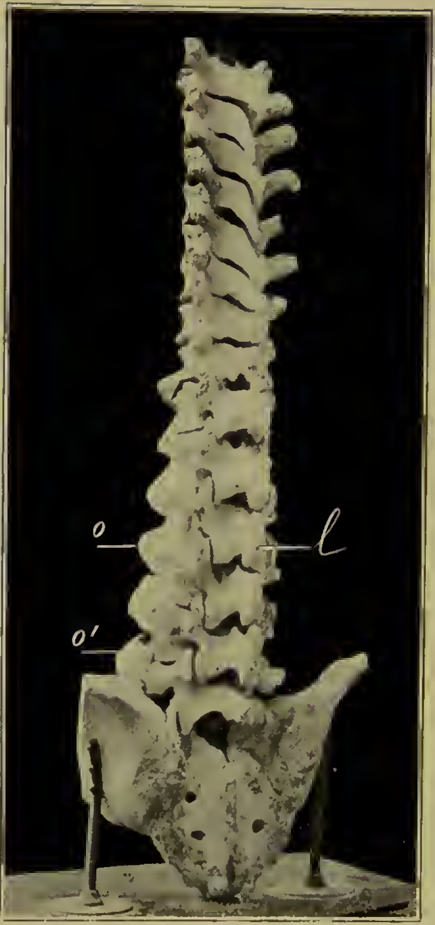


Fig. A.

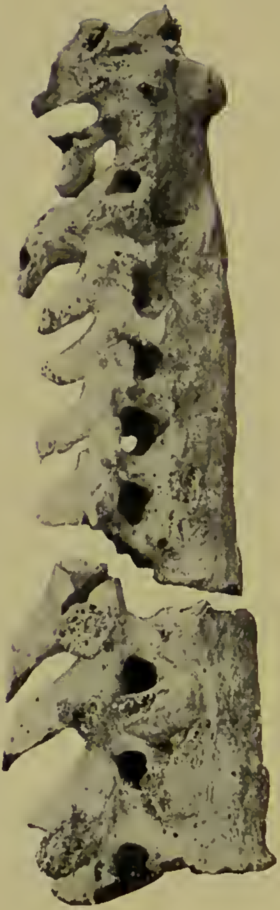


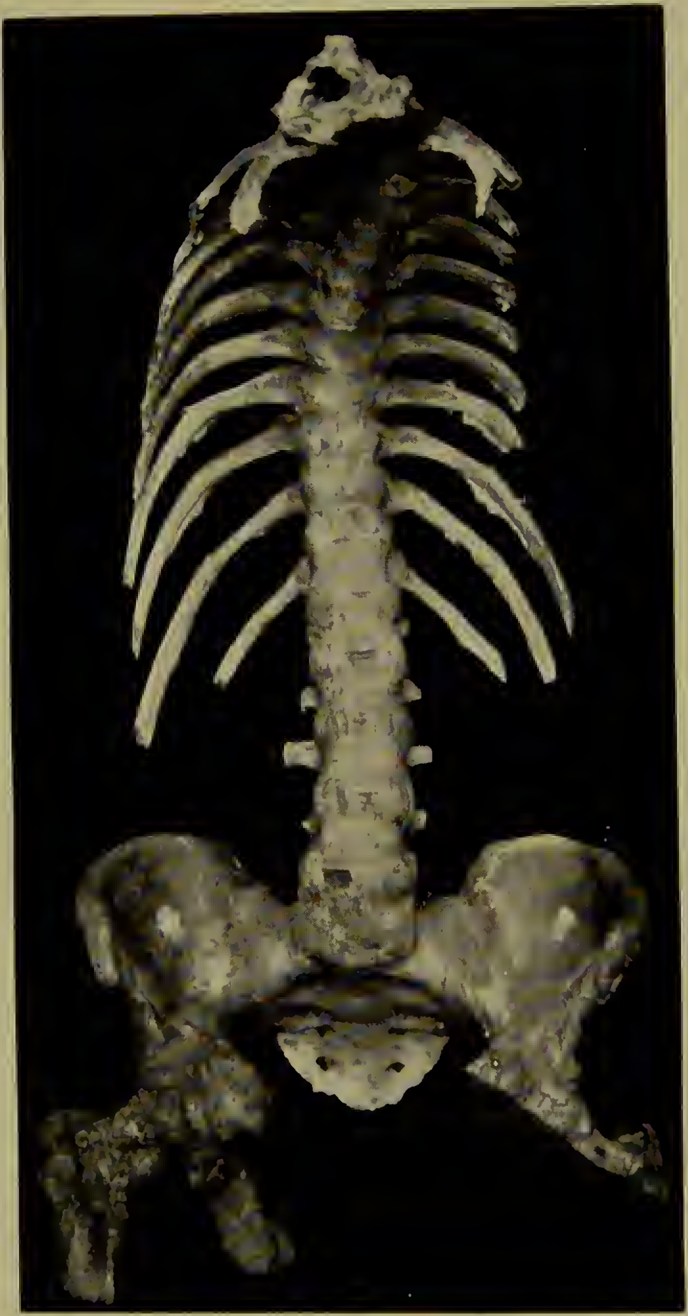
Fig. B.

Fig. A.—Chronic Vertebral Rheumatism with complete ankylosis (from Prof. Teissier of Lyons' collection).

Note the uprightness of the spinal column, also the presence of enormous osteophytic outgrowths *O*, *O′*, with marked enlargement of the spinous processes at *l*, and narrowing of the intervertebral foramina. The patient had perhaps suffered from syphilis.

Fig. B.—Chronic Rheumatism of the Cervical Region (from Prof. Teissier's collection). Note here also the uprightness of the spinal column, the complete ossification of the ligaments uniting the various segments, the enormous bony enlargement of the spinous process of the third cervical vertebra, the very pronounced narrowing of the intervertebral foramina.

PLATE XXXIII.



Spondylose Rhizomélique.—(In this case there is a fracture of the cervical column in addition to the "spondylose"). Note the smooth ossification of some parts of the anterior spinal ligament or of the discs in the dorso-lumbar region, the absence of osteophytes, the ankylosis of the ribs to the vertebræ, the subsidence of the spine into the pelvis, the tilt of the sacrum, the complete ankylosis of the hips formed by the ossification of the ligaments surrounding the joint.

(Plate published by Dr. André Leri, in "Review of Neurology and Psychiatry," Edinburgh).

PLATE XXXIV.

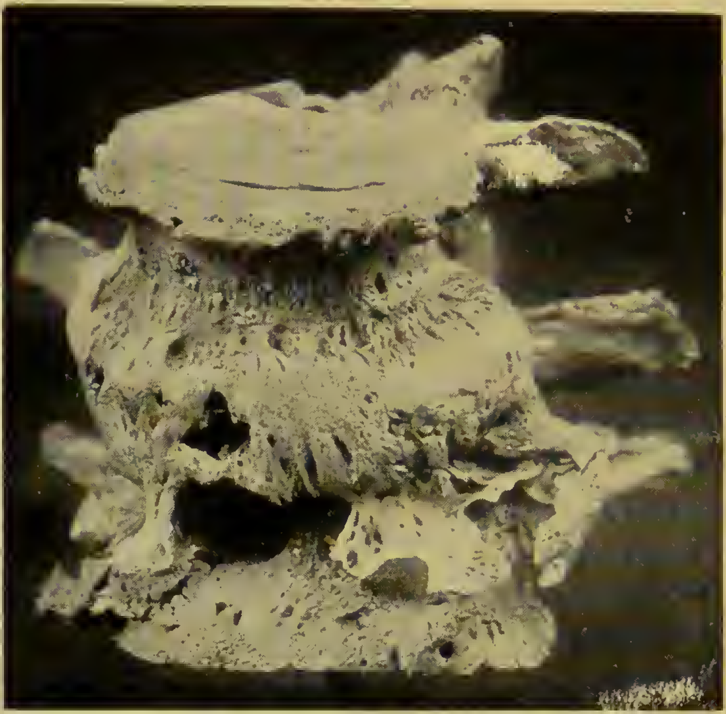


Fig. 1.—"Spinal Rheumatism."—Note the numerous and irregular osteophytes, the voluminous protuberances, especially at the level of the disc. (Musée Dupuytren).

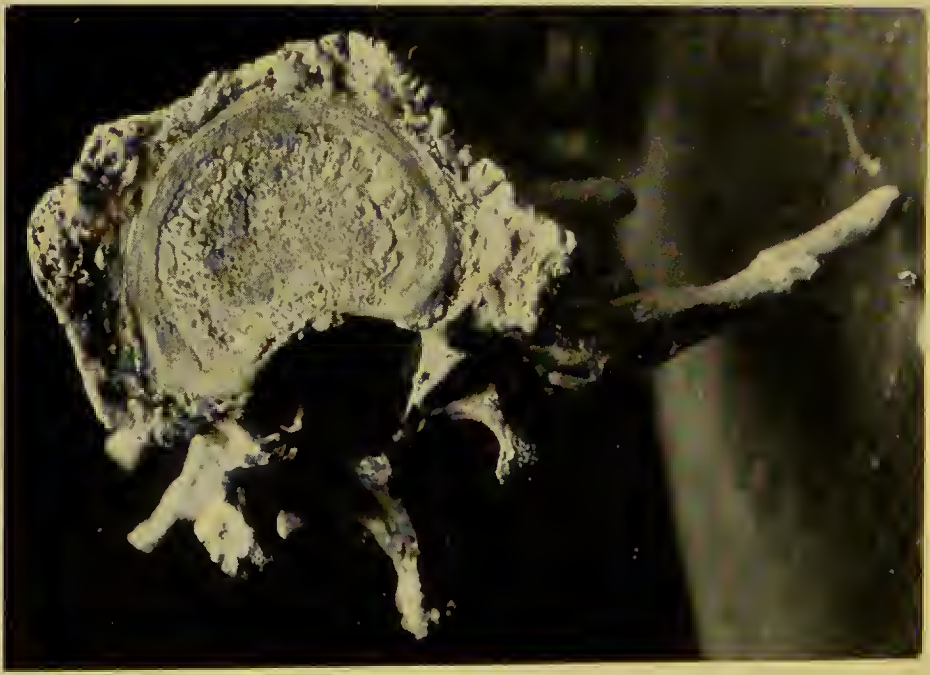


Fig 2.—A Vertebra, seen from above, in a case of "Spinal Rheumatism." Note the development of the osteophytes which surround the vertebra and almost double its surface

The anatomical changes shown in the above figures, like those in *Plate XXXII*, are typical of osteo-arthritis of the vertebral column. Compare the rough nodular character of the bony formations in these plates with the smooth regular ossification shown in *Plates XXVIII*, *XXX*, and *XXXIII*.

(Plate published by Dr. André Leri, in "Review of Neurology and Psychiatry." Edinburgh

Chapter XIX.

MORBID ANATOMY OF SPONDYLITIS DEFORMANS.

The pathological changes may invade the whole or only a portion of the spinal column, and indeed may even be confined to one segment of it. In some cases the bony and articular surfaces are more particularly affected, in others the ligamentary and capsular structures chiefly suffer.

Briefly considered, the chief anatomical characters are as follows :—

The intervertebral discs undergo fibrillation and absorption to a greater or less extent, and in some cases are entirely replaced by bone, thus giving rise, in rare instances, to a condition of true bony ankylosis of the bodies of the vertebræ.

The lateral aspects of the vertebral bodies become the seat of new bony outgrowths, which, fusing with similar formations on neighbouring segments, assume a somewhat stalactitic form, uniting the vertebræ one to another, so to speak, like a bony clasp. The various conditions are well exhibited in *Plates XXIX to XXXII.*

Usually these longitudinal osseous bands are placed symmetrically, covering, according to their width, more or less of the space between the transverse processes and the edge of the anterior common ligament ; they may, however, be only present on one side.

According to Koehler,[1] in some cases the bodies of the vertebræ may undergo marked atrophy of their substance, and the morbid change, being often more marked in one portion of the segment than another, leads to pronounced alterations in the shape of the affected vertebræ, as regards height, depth, and breadth, and thus the resulting curvature may be extremely irregular in contour.

The cartilages of the intervertebral and costovertebral articulations undergo fibrillary changes and erosion, the resulting ivory-like surface often showing grooves running along the line of direction of the chief movements of the joints. Bony lipping and osteophytic outgrowths ensue, and the enlargement of the superior and inferior oblique processes may encroach to a marked degree upon the spinal canal and intervertebral foramina. Not infrequently the odontoid process becomes greatly hypertrophied, projecting upwards towards the foramen magnum to the extent of half an inch beyond its ordinary level.

Sir William Gowers[2] quotes Solbrigg as having noted a case in which the nerves issuing from the medulla were interfered with by such exostotic outgrowths. At the same time the capsular and ligamentary structures undergo bony transformation to a variable degree.

In both the intervertebral and costovertebral articulations, not only may the articular surfaces be firmly welded together, but the capsules also become ossified. Similarly, extensive ossification of the spinal ligaments takes place, not only of those in more intimate relation with the column, but also of those uniting the various spines of the vertebræ; indeed the ossification of the various ligaments may proceed to such an extent as almost to encircle the bodies and intervertebral discs, but these latter notwithstanding may remain intact within the bony casing.

While the foregoing account may suffice as a general description of the morbid anatomy of the condition, it is necessary now to allude in detail to those characteristic of the types isolated by Strumpell,[3] and Marie,[4] and von Bechterew.[5]

Marie's first study of the pathological anatomy of spondylose rhizomélique related to a skeleton in the Musée Dupuytren, in which he noted ankylosis of the lumbar vertebræ and hip joints, both being the seat of marked proliferative changes. The complete rigidity of the spine in this case was attributed by Marie to the ossification of the anterior common and costovertebral ligaments.

According to Marie and Leri[6] the most remarkable feature of this group is the almost complete ossification of the ligaments of the spinal column, the articular processes, on the other hand, being only slightly affected: such local changes as may take place in the intervertebral joints being confined to the periphery of the opposing articular surfaces and the enclosing capsule. The ligamenta flava, as well as the costovertebral ligaments, show bony transformation, especially noticeable in their superior and inferior fasciculi, while on the other hand the smaller bundles attached to the intervertebral discs escape.

The costo-transverse and interspinous ligaments are involved, the latter forming flying buttresses of bone which stretch from spine to spine along the posterior aspect of the vertebræ.

The same changes are met with on the anterior surface of the vertebræ, but the bony change affects the intervertebral disc rather than the anterior common ligament, which, like the posterior, may enjoy comparative immunity. The spinal cord and intervertebral foramina preserve their normal calibre, not being encroached upon by any outgrowths.

The same process obtains in the hip and shoulder joints, and may also involve the sterno-clavicular and temporo-maxillary articulations, the capsular and ligamentary structures being affected rather than the articular surfaces, the whole morbid condition, to use the nomenclature of Leri,[6] corresponding to "une menisco ligamentite ossificante."

Raymond and Babboneix[7] have reported a case of spondylose rhizomélique in which the patient, contrary to the rule, was of the female sex. An autopsy was made, and was remarkable for the variety of the morbid changes found, apart from those concerned with the skeletal structures.

The authors give a detailed account, the result of which they summarize as follows: (1) Changes in those glands associated with internal secretion: ovarian hypoplasia, complete hypoplasia of the follicles of Malpighi of the spleen, intense multiple lesions of the thyroid body, with slight lesions of the pancreas, suprarenal glands, and pituitary body; (2) Thoracic lesions consisting for the most part of perivisceritis and hypoplasia of the aorta; (3) Purely fibrous ankylosis of the joints at the roots of the extremities, mixed ankylosis, fibrous and osseous, of the vertebral column. They suggest that the changes in the internal secretions resulting from structural alterations in these various glands, widespread as they were, had given rise to disturbances of the general nutrition to which all the perivisceral and peri-articular changes, as well as the osseous ankylosis, might be ascribed; they further suggest that the structural changes in the glands may have resulted from the various infections—measles, scarlatina, small-pox—from which the patient had suffered in rapid succession during early childhood.

In the only case of von Bechterew's[5] series which came to autopsy, the following changes were noted :—

In the upper dorsal region, the intervertebral discs had undergone a variable degree of atrophy, in some cases complete absorption had taken place, while the bodies of the vertebræ were fused together to a greater or less extent, and osteophytic outgrowths were present on their surfaces. The spinal canal and intervertebral foramina were of normal calibre, and the intervertebral articulations were free from disease.

In the cervical and upper thoracic regions the spinal meninges were the seat of chronic inflammatory changes, mainly localized to the pial and arachnoid membranes ; the thickening was most noticeable on the posterior surface of the cord in the upper thoracic region.

The posterior roots, likewise the anterior in lesser degree, were found to have undergone degeneration, the changes being more marked in those of the cervical and thoracic region than in those situated at lower levels.

The columns of Gall and Burdach—especially the former—showed degeneration in their cervical and upper dorsal segments, this being secondary to the root degeneration, which in turn was ascribed to changes in the spinal ganglia, these being adherent to the dura mater. These changes were not confined to the posterior columns, the anterior being also affected.

It is interesting here to note that as far back as 1874, Rotter,[8] according to Garrod, had observed degeneration of the spinal cord in a case of slight cervical spondylitis involving the occipito-atlantal joints.

Leri[6] also has published the post-mortem findings in a case of " la cyphose hérédo-traumatique," which, as before stated, he has identified with the von Bechterew type. This authority regards the condition anatomically as one of kyphosis with marked ossification of the spinal ligaments, the bony change taking place chiefly in the ligamenta flava and the anterior common ligaments, the latter structure in his case being transformed into a moniliform osseous band extending from the sixth to the twelfth dorsal vertebræ, the bony transformation of the ligamenta flava being also strictly limited to the same portion of the column.

The fact that all these ossifying processes were localized to one and the same region of the column is adduced by Leri as a strong argument in favour of the traumatic origin of this group of cases. He supports his contention by pointing out the similarity obtaining between the character and distribution of these morbid changes and those observed by him in the spinal column of a man in whom one of the bodies of the vertebræ had been penetrated by a bullet, the same localized ossification of the anterior common ligament having taken place. This authority had also noted a similar bony transformation in sequence to laminectomy.

Leri, as a result of his study of the anatomical appearances found in spondylitis deformans, believes it possible to distinguish two chief varieties of vertebral ankylosis, one in which the rigidity is mainly due to ossification of the ligamentary and capsular structures, and one in which the bony outgrowths and articular changes are chiefly responsible for the immobility. Of the first variety he recognizes two types: a generalized form, of which spondylose rhizomélique is the example, and a localized represented by "la cyphose hérédo-traumatique," or the von Bechterew type.

Under the second variety are placed cases of spondylitis showing the following characteristics: Consolidation *en masse* of the bodies of the vertebræ with ossification of the intervertebral discs, the column often losing its normal curves, and becoming rigidly fixed in a vertical position, in this respect contrasting with the kyphosis found in the other variety; hypertrophy of the spinous processes, with little or no ossification of the supraspinal ligaments, relatively little involvement of the intervertebral articulations, but voluminous formation of osteophytic outgrowths, with marked encroachment upon the intervertebral foramina.

In addition, the following important pathological distinction is made between the two main groups, namely, that in the former, or what may be termed the ligamentary type, the bony changes in the vertebræ are predominantly of a rarefying or atrophic character, while condensation of bone—osteoplastic osteitis—more particularly distinguishes the latter.

The ossification of the ligaments, so characteristic a feature of the first group, is held to be secondary to the primary rarefying changes in the bony structures, and is regarded by Marie and Leri as being of a compensatory nature, in that its incidence imparts rigidity to the otherwise continuously yielding column, limits the degree of deformity, ensures rest, and promotes cure.

We have now to consider two examples of Janssen[9] in which complete rigidity of the spine arose as part of a generalized rheumatoid arthritis implicating in the one case both hips, knees, and tarsal articulations; in the other all the peripheral joints had undergone gradual stiffening.

Here again we find, as in spondylose rhizomélique and the von Bechterew type, the bony changes to be purely atrophic in character.

In addition, great fragility of the bones was noted. Both the cancellous and the compact bony tissues were the seat of rarefactive changes. Nowhere were there to be found any osteophytic outgrowths. The cartilages had undergone atrophy.

Janssen considered that the condition should be sharply differentiated from osteo-arthritis.

In conclusion it would appear therefore that in this, so to speak, vertebral, as well as in the peripheral type of arthritis deformans, there are indications of the same great cleavage into the rheumatoid or atrophic and the osteo-arthritic or hypertrophic divisions. Under the former will be grouped on this pathological basis the von Bechterew type, the spondylose rhizomélique of Strumpell and Marie, and also those cases of spinal rigidity arising as part of a generalized rheumatoid arthritis, and under the latter of course will come the osteo-arthritic variety.

According to the modern view upheld by Osler and other authorities, the von Bechterew and Strumpell-Marie types are not to be regarded as distinct morbid entities, but as merely varieties of the vertebral type of arthritis deformans. Should Leri's observations as to the essentially atrophic character of the bony changes in these two groups be confirmed, it would go far to establish the existence of a rheumatoid as opposed to an osteo-arthritic type of spondylitis deformans.

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Chapter XX.

CLINICAL ACCOUNT.

MODES OF ONSET.

In some rare instances the malady begins more or less suddenly, pain and stiffness of some portion of the column supervening quickly after some traumatism, or may be as a result of exposure to cold or damp. We have before alluded to this comparatively rapid mode of onset after trauma, more especially in relation to the von Bechterew variety, or la cyphose hérédo-traumatique, but it must not be inferred that such is peculiar to this type, as it has been noted occasionally in all forms of spondylitis.

Again, cases may ensue almost as abruptly after exposure to cold and damp; thus Popoff[1] narrates an instance resembling von Bechterew's group, in which a young fisherman after five hours' wading suffered the same evening with pain in loins, knees, and spine, developing within two months marked rigidity of the column. In a case which came under my own observation symptoms of the affection manifested themselves almost directly after exposure during a severe thunderstorm. It is moreover possible that in this particular instance mental shock played a part in its genesis, inasmuch as the subject, a cowherd, while sheltering with some of his cattle beneath a tree, witnessed the destruction by lightning of several of the beasts in his immediate vicinity. Previously healthy, the patient took to his bed with what was deemed to be an attack of "rheumatism," and when seen by me a twelvemonth afterwards his spinal column had become rigid throughout its whole length.

Goldthwait,[2] in his article on "Osteo-arthritis of the Spine," tells of a woman fifty-seven years of age who, after being soaked through in a thundershower, was seized the next day with pain in the neck, which rapidly increased in severity, any attempt at movement of the cervical spine causing intense suffering. This acute phase lasted for about two months, during which period a condition of paresis almost amounting to paralysis of the left arm developed, with a less marked but notable degree of weakness in the right arm and both lower extremities. When seen thirteen months after the onset, there was present marked rigidity, especially of the cervical portion of the column, with thickening, apparently of bony nature, over the transverse processes of the vertebræ of this region ; the paresis of the lower extremities and right arm had completely disappeared, but there was still marked weakness of the left arm.

This same observer narrates an instance in which osteo-arthritis of the spine developed after a "nervous breakdown," the rigidity and kyphosis of the upper dorsal and cervical column being subsequently complicated by numbness of the upper extremities, with marked atrophy of muscles of chest, shoulder, and upper back.

In the majority of instances, however, the condition of spinal rigidity is only arrived at very slowly and insidiously, and not infrequently its presence is overlooked, owing to the fact that the pains are often referred to the extremities rather than to the affected portion of the column.

Thus paroxysms of cervico-occipital and brachial neuralgia may recur again and again, without exciting the remotest suspicion as to the true nature of the affection, any limitation of the movements of the column that may be present being thought to be amply accounted for by muscular spasm. Undoubtedly, during the progress of these painful paroxysms a high degree of muscular spasm often prevails, and that such contributes in no small degree to the fixity or stiffness of the column is shown by the marked increase in mobility which follows their subsidence, but nevertheless, even at this early stage, in the interparoxysmal period some degree of synovial creaking may be elicited either by voluntary or passive movements, thus affording a valuable aid to diagnosis. Its importance as a clue is enhanced in that it may be present before any appreciable degree of permanent limitation in the movements of the affected portion of the spine has resulted.

In some cases, in addition to the cervico-occipital neuralgia, paræsthesiæ and pains of variable intensity may be experienced along the upper limbs.

In others, pains of a gripping or girdle-like character may be present in disease of the thoracic region of spine, or stiffness, and pain of the lumbar muscles of unilateral or bilateral distribution, may for long mask a slowly progressing ankylosis of the lumbar column.

In the lower limbs similar pains and paræsthesiæ may be experienced, or symptoms more of a motor type, such as trembling, weakness, or stiffness of the muscles, may be complained of. Such patients find a difficulty in rising from their chairs, or an unusual effort is required to cross one leg over the other.

Forestier,[3] of Aix-les-Bains, lays great stress on what he terms the "pseudo-neuralgic" type of spondylitis, whose onset is marked by severe pain, either localized in the column or referred to the limbs, where it may follow a unilateral or bilateral distribution. These pains, he observes, give rise to considerable muscular spasm and stiffness of the vertebral column. After a variable period the pains subside, and rigidity of the column becomes the prominent symptom. According to this observer, Leyden,[4] Homolle, and others have noted this period of pseudo-neuralgic manifestations in spondylitis deformans.

Forestier believes that in this pseudo-neuralgic variety the disease is mainly situated in the synovial joints of the vertebræ, from which sites inflammatory changes spread to the meninges and nerve roots. The subjects of this form of spondylitis, in his experience, have usually suffered previously from rheumatic manifestations, even definite attacks of polyarthritis of the limbs, or more rarely gout or gonorrhœa.

Warrington[5] also reports a case of rigidity of the spine in which swellings of the knees, ankles, and small joints were present; these latter articulations recovered, but the immobility of the column was apparently of permanent character.

In this case the affection began with tight, gripping pains across the lower part of the thorax, of persistent character and intensified by movement. These lasted for two years before the stiffness of neck and back became marked. The disease in the peripheral joints being evidently synovial in site, this observer considered it legitimate to infer that the spinal rigidity was due to similar change in the synovial structures of the intervertebral articulations.

The varieties of spondylitis in which the vertebral rigidity is preceded by these neuralgic pains are those of the von Bechterew type or la cyphose hérédo-traumatique, and also, according to Marie, those classed as spondylose rhizomélique; I may add that in my experience these premonitory neuralgias are extremely common antecedents of spondylitis arising in the course of rheumatoid arthritis.

It is interesting here to recollect that the bony changes in the former varieties are also predominantly of an atrophic nature.

On the other hand, when we come to deal with the hypertrophic or osteo-arthritic form of spondylitis, we find that in the initial stages pain, if complained of, is localized strictly to the affected portion of the column, and only at a later period do the limbs or trunk become the seat of referred pains following pressure upon nerve roots by exostotic outgrowths encroaching upon the intervertebral foramina.

Not infrequently the spinal rigidity takes place so insidiously as to be entirely overlooked, and only when the subsequent pains, by their chronicity and severity, lead to careful examination of the spinal column, is the true nature of the affection recognized. For example, a chronic sciatica for long ascribed to constitutional causes such as gout and rheumatism may find its explanation in an osteo-arthritis of the lumbar column.

CLINICAL APPEARANCES.

The most striking or central feature of spondylitis deformans is the rigidity, combined with a greater or less degree of deformity of the spinal column. This rigidity may be generalized, in severe cases the whole of the spine from the occiput to the sacrum undergoing a fusion *en masse*, all varieties of motion, whether extension, flexion, or rotation, being effectually abolished. On the other hand, the immobility may be limited to the lumbar, dorsal, or cervical region respectively, or it may be even restricted to two adjacent vertebræ; thus Gowers notes that the atlanto-occipital joint may alone show signs of disease.

Adams was of opinion that the cervical and lumbar regions were more frequently affected than the dorsal portion of the column.

As regards deformity, the most marked change is an exaggeration of the physiological dorsal curve, with very commonly a disappearance of the normal lumbar curve: in other words, a dorsal kyphosis without a compensatory lordosis; but in severe cases the whole spine may be curved with its convexity backwards.

On the other hand, there may be present no bowing of the spine, the vertebral column being perfectly straight and rigid —the so-called "poker-back,"—the normal curves being obliterated.

It is possible, however, for extensive rigidity to be present without any change in the normal curves of the spine, as exemplified in one of Goldthwait's cases of osteo-arthritis. The contour of the curvature is as a rule smooth, and free from any localized projection of the vertebræ or angularity, but according to Koehler, in some cases the spinal curvature may be strikingly irregular. Though the curvature is as a rule antero-posterior in direction, there may be a slight lateral list of the column to one or other side, especially in the cervical and dorsal regions. Should the costo-vertebral articulations be involved, as happens in the severer cases, there will be flattening of the chest in an antero-posterior direction, with immobility of the ribs, leading to the breathing being almost wholly abdominal in character. Rotation or twisting of the vertebræ may also be present, leading to projection of the ribs on one or other side.

The joints of the limbs are implicated to a variable extent, more commonly those of the shoulder and hip. The movements of the former are but slightly interfered with, the patient at most complaining of a slight difficulty in abducting and elevating the arm; on the other hand, in the case of the hip-joint, the results of the morbid changes are much more marked, leading as a rule to fixation of the thigh in a position of flexion, adduction, and external rotation; more rarely the hip joint may be ankylosed in a position of extension.

With regard to the other joints, the knees are occasionally affected, and they may assume a position of permanent semiflexion. In addition, the temporo-maxillary and sterno-clavicular articulations may be involved, and in rare instances the small joints of the hands and feet.

In concluding this brief account of the clinical aspect of these cases, it is hardly necessary to add that the character and degree of the spinal deformity will naturally vary according to the seat and extent of the disease, likewise with the course pursued by the morbid process attacking the vertebræ, whether slow or rapid. It is obvious, for example, that if such proceed more swiftly on one side of the vertebral bodies, it will lead to a lateral list of the column; this is more commonly to be seen in the cervical and dorsal regions of the spine.

Such in brief are the alterations in the spinal column and joint structures generally. It is now necessary to pass on to the other features of the malady, and these are more conveniently discussed under two headings, namely, those referable to some interference with the motor functions of the body and those due to sensory disturbance.

We propose to deal first with those pertaining to the sensory sphere. While, as we have seen, the onset of the malady may be marked by various paræsthesiæ, tingling, numbness, or radiating pains, these sensory disturbances are by no means confined to this initial period, as even after the spinal rigidity is well established they may continue to manifest themselves.

Cases of spondylitis deformans, however, present great diversity in this respect. In some instances the sensory modifications are insignificant or wholly absent, in others widespread and severe. Thus Goldthwait has noted that in the subjects of "osteo-arthritis" of the spine "numbness" may be complained of throughout the whole extent of one or other of the limbs.

In others persistent hyperæsthesia, involving either the whole or a portion of the limb, may be a feature, or some area of skin over the trunk may be similarly affected.

Commonly these sensory perversions are unilateral, and when symmetrical in distribution one side is affected before the other. Pain in all varieties of spondylitis is an extremely variable symptom, and what is somewhat remarkable, both pain and other sensory aberrations are not necessarily associated with or dependent upon pressure by exostoses, inasmuch as these have been prominent symptoms in cases where an autopsy has revealed the absence of any encroachment upon the intervertebral foramina.

It is probable, however, that localized meningitis exists more commonly than is thought, giving rise to irritation or compression of nerve roots.

Oppenheim,[6] in his account of spondylitis, notes the occurrence of brachial, intercostal, and crural neuralgias, and to my former statement that cervico-occipital neuralgia is a very constant and early symptom of cervical spondylitis I would add that it may or may not be accompanied by similar radiating pains along the upper limbs. Paroxysms of this cervico-brachial neuralgia may recur again and again even in long-standing cases.

Going more into detail in regard to this cervico-occipital neuralgia, one notes that during an attack the cervical vertebræ are very sensitive to percussion, and from the spine pains variously described as shooting, stinging, etc., pass upwards to the occipito-parietal region of the scalp, and at the same time, in many instances, sweeping outwards over the trapezoidal region of the shoulder.

These pains may be uni- or bilateral, but are usually more severe on one side, and are followed by objective hyperæsthesia and hyperalgesia of the skin over the painful area. These areas of tenderness vary in extent in different cases ; in some only the skin over the nape of the neck may be affected, but in others the hyperæsthesia extends over the occipito-parietal region on the same side. The tenderness may be exquisite, women, for instance, being unable to bear the insertion of hairpins or any tension upon the scalp. With the decline of a paroxysm, profuse sweating takes place over the affected area, and the previous hyperæsthesia is not infrequently replaced by temporary dulling of sensation, especially as regards pain-stimuli. With remissions, these paroxysms may last for days, and their frequent repetition is followed by premature greyness and thinning of the hair. In some instances the area of tenderness may extend from the edge of the hairy scalp over the nape of the neck, and outwards along the suprascapular region to the shoulder, and in some cases may be prolonged forwards and downwards over the side of the neck to the subclavicular region.

When the cervico-dorsal region is affected, brachial and intercostal neuralgia may add their quota to the patient's suffering, and when in such cases the skin over the front of the upper chest has been hyperæsthetic, I have found the ciliospinal reflex much brisker than on the unaffected side. In addition, when the skin over one side of the chest posteriorly has been similarly affected, the superficial scapular reflex may be notably brisker than on the opposite side.

It is here convenient to remember that Gowers[7] has pointed out that there may be ocular symptoms pointing to disturbance of the sympathetic in this disease. Jouve, quoted by Teissier,[8] has also remarked this occasional coincidence of oculo-pupillary changes.

Girdle pains or gripping sensations of varying intensity may be experienced when the lower thoracic or lumbar bodies are implicated. Such may be spontaneous, or only evoked by some particular movement.

Also, in disease of this region, radiating pains in the lower extremities may cause great distress. We have before alluded to the sciatic pains that may follow the formation of exostotic outgrowths in the lower portion of the column, but they have been noted also in cases of chronic spinal rigidity unattended by bony nodes.

According to Gowers, occasionally a descending neuritis may result from severe pressure on the nerve roots by exostoses.

Areas of permanent anæsthesia have been met with in various parts of the trunk and limbs, usually in sequence to severe pain in the same area. Allen[9] has noted anæsthesia over the occipital region in a case of cervical spondylitis. Similar areas of diminished sensation were observed by von Bechterew along the tracks of lower thoracic, cervical, and sometimes of lumbar, nerves. I have seen in rare instances such areas of anæsthesia in the arm and leg which appeared to follow a root rather than a peripheral nerve distribution.

In reviewing the literature on this subject of sensory disturbance in spondylitis deformans, one is led to the conclusion that disorders of sensation occur much more frequently than is commonly considered, and I quite agree with McCrae[10] that more detailed examination of these cases from this point of view is indicated. Garrod[11] also is inclined to think that the sensory disturbances in this malady have not received the attention they deserve.

Passing now to the consideration of disorders of the motor system. In the early stages signs of irritation predominate, and a high degree of spasm, or " stiffness," as patients term it, prevails, and doubtless at this period much of the fixity of the spine is due to this tonic contraction of the muscles. Such spasms may be spontaneous in origin, or may only be evoked by certain movements, and when present may give rise to intense suffering. Spasmodic torticollis has been noted in spondylitis of the cervical column. Similarly in the upper dorsal region marked spasm of the muscles of the shoulder girdle and neighbouring spine have been described. The spasms may affect the muscles of the limbs, and in one recorded instance gave rise to a certain spasticity of gait.

Rigidity of the abdominal muscles has been noted by Dercum[12] in a case of spondylose rhizomélique, and was attended by excessive hyperæsthesia of the abdomen. Muscular wasting may or may not be present, and when it exists is variable, both as regards its degree and distribution. In some cases it is tonic in character, in others the musculature generally is flabby.

It may be generalized or localized ; in the case of the former such widespread muscular atrophy is not necessarily attended by pain, or any other form of sensory disturbance, and it appears indifferently throughout all forms of spondylitis, whether attended by exostoses or not.

C. S. Potts[13] records a case of spinal rigidity with excessive atrophy of the muscles of shoulder, and also atrophy, not so excessive, of all other muscles. In addition, myotatic irritability was increased and fibrillary muscular contractions were noticed. The deep tendon reflexes were increased in both upper and lower extremities ; ankle clonus also was present, likewise an increased jaw-jerk and Babinski's sign. The general symptomatology of the case led this observer to suggest that but for the spinal rigidity the condition presented a striking general resemblance to that of amyotrophic lateral sclerosis. I have also reported a case of rigidity of spine in which such widespread muscular atrophy was present, with marked exaggeration of reflexes of both upper and lower extremities.

In many cases, however, apparently identical as regards the skeletal changes, muscular atrophy is slight and confined to the muscles of the shoulder, such as the supra- and infra-spinati. Oppenheim points out that atrophy may be present in the muscles of the extremities, and Warrington records a case of chronic rigidity of the spine, in which the atrophy was confined to the supra- and infra-spinati, together with those of the thenar and hypothenar eminences and first dorsal interosseous muscle.

Schultze, quoted by Oppenheim, has recorded a case of spondylitis deformans associated with pseudo-hypertrophy of the muscles.

Weakness of the spinal muscles, or of those of the extremities, may be present without any noticeable degree of atrophy of the affected muscles. It may be variable in degree and distribution, more frequently affecting one or other of the extremities, and is often preceded by numbness in the limb.

Oppenheim notes the occurrence of atrophy with partial paralysis of the extremities. Goldthwait, referring strictly to osteo-arthritis of spine, has noted that when the nerve-roots of limbs are compressed, neuralgic pains followed by paralysis may result. The palsy, he states, may present all grades of severity, from slight paresis up to complete loss of voluntary movement.

In continuation, he points out that such palsy is usually unilateral, rarely bilateral, and in the case of the latter the extent varies in the two limbs, which are not concomitantly involved, the paralysis as a rule developing first in one limb and then spreading to the other.

The paralysis is usually peripheral in type; it sometimes happens, however, that when the disease pursues a very rapid course osseous deposit takes place in the posterior common ligament, and may encroach so markedly upon the spinal canal as to cause a compression paraplegia. This observer also notes that these two types of palsy, peripheral and central, may be present in the same patient, that is to say, peripheral palsy in the upper limbs from compression on the nerve roots, and paraplegia of the lower extremities from direct compression of the spinal cord by exostoses.

The reflexes, both superficial and deep, in all varieties of spondylitis, show extreme variability. They may be normal, exaggerated, or absent, and the corresponding reactions, whether deep or superficial, in the same patient, may be in striking contrast. In reviewing the cases reported in the literature, the general tendency appears to be towards exaggeration of the deep reflexes in the lower extremities, but solitary cases have been recorded in which the deep reflexes in both upper and lower extremities were exaggerated. Double ankle clonus may be present, and Babinski's sign or extensor-toe-response has been noted by Warrington in a case which he considered to approximate to the pseudo-neuralgic type of Forestier. Kernig's sign was also noted by the same observer.

No change in the electrical reactions has been noted, with the exception of those cases in which, as the result of compression of nerve roots, either by localized meningitis or exostoses, atrophy, with reaction of degeneration, has occasionally been noted.

As regards other symptoms, I recorded some time ago a case of spondylitis deformans presenting a cervico-dorsal kyphosis with permanent rigidity of the affected portion of the spine, in which there were present very marked vasomotor phenomena of the Raynaud-like type affecting face, extremities, and genital organs.

There were present also in this case considerable induration and thickening of the muscles of the right side of neck, with permanent rotation of the head and neck to the opposite side. It is convenient here to record that such thickening in the muscles of the cervical region is usually unilateral. I have, however, seen great induration and thickening on both sides of the neck, leading to greatly increased width of the nuchal region.

Difficulty of deglutition has been noted in several cases. In some instances it has appeared to be due to the presence of exostoses projecting into the pharynx, and such have been recognized during life by buccal palpation by Koehler[14] and others.

Diminution of the sexual functions has been noted, but, as far as I am aware, no case of marked interference with the vesical or anal sphincters has been recorded. As regards visceral complications, albuminuria deserves mention. Tuberculosis also has been met with, and should be borne in mind in view of the important etiological rôle ascribed to this factor by the French authorities.

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CHAPTER XXI.

TYPES.

WHILE the foregoing remarks may suffice for a description in general of the cases classed together as spondylitis deformans, it is necessary here to allude briefly to the special characteristics of certain suggested types, for example, the von Bechterew[1] type, or la cyphose hérédo-traumatique of Marie and Astie,[2] the variety known as spondylose rhizomélique of Strumpell[3] and Marie,[4] that due to osteo-arthritis of the spine, and finally the one due to rheumatoid arthritis of the spine.

Briefly described, the following are the characteristics (*Plate XXXV, Fig. C*) of the *von Bechterew type*:—

1. It occurs in both sexes.
2. Immobility of the whole or portion of the spine; no pain on percussion of spine.
3. Kyphosis of the lower cervical and upper thoracic vertebræ without compensatory lordosis; displacement of the head downwards and forwards; half-flexed and prominent knees when patient is in a standing position; flattening of the thorax, with breathing almost wholly abdominal.
4. Paresis of muscles of trunk, neck, and extremities, with slight wasting of spinal and scapular muscles.
5. Spasmodic twitching and contracture of the muscles of the extremities owing to irritation of nerve roots; reaction of degeneration in advanced cases.
6. Paræsthesiæ, diminished sensation, and radiating pains, especially in those areas supplied by the cutaneous branches of the thoracic, lower cervical, and occasionally lumbar nerves.
7. Exaggerated tendon reflexes in the lower extremities.
8. Joints of extremities not affected.

The following are the characteristics of the type *la spondylose rhizomélique,* according to Marie and Leri's graphic account:—

1. Develops almost exclusively in men, but isolated cases

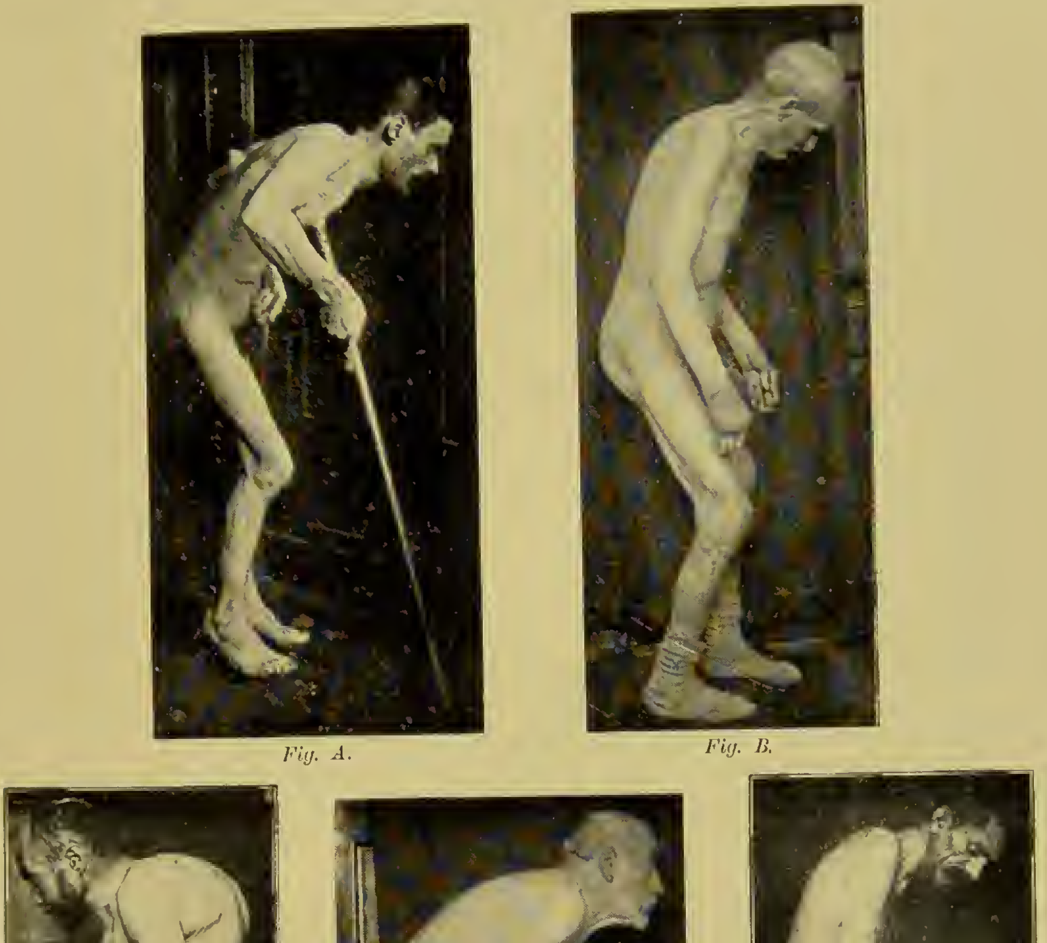


Fig. A.

Fig. B.

Fig. C.

Fig. D.

Fig. E.

Fig. A.—Spondylose Rhizomélique, "flexion type." Note the flexed position of the hip joints.

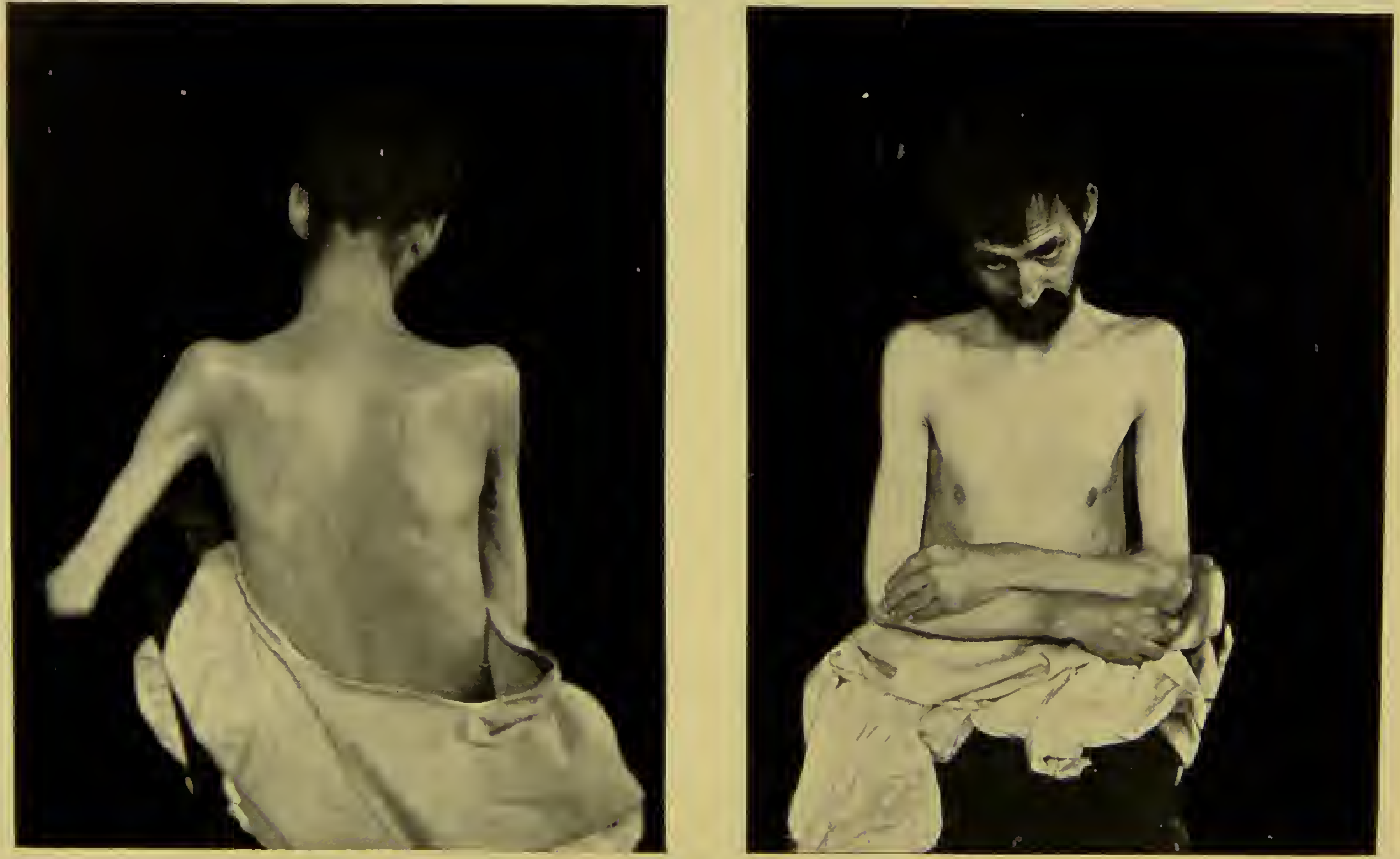
Fig. B.—Chronic Ankylosing Vertebral Rheumatism, with commencing ankylosis of the hip joints. The deformities of the hands and the slight relative curvature of the vertebral column are to be noted.

Fig. C.—Hereditary Traumatic Kyphosis. Note the extreme degree of curvature limited to the dorsal region. (Patient of Marie and Astié).

Fig. D.—Duplicature Champêtre, or Peasant's Stoop of Marie. Note the kyphosis in the lumbar region and the stooping attitude.

Fig. E.—Spondylose Rhizomélique, "extension type," with in addition luxation of the cervical column. Note the slight curvature of the spinal column, the flattening of the thorax, and the normal state of the extremities.

PLATE XXXVI.



Cervico-dorsal Rigidity of Spine in rheumatoid arthritis, with involvement of both shoulders, elbows, wrists, also hips, knees, and ankles. Head permanently bent forwards, with slight deviation to right, widespread atrophy of trunk and limb muscles.

have been recorded in women. I have seen one example of the disease in a young woman aged twenty-three.

2. Disease begins with acute pains, preceding may be for a long period the ankylosis, which commences in the coccygeal, sacral, and lumbar regions of the column, afterwards extending to the dorsal and cervical region, in which also we get a similar preliminary neuralgic stage; according to Teissier, the disease may commence in the coxo-femoral joints.

3. In a typical case there is ankylosis of the spinal column, also of hip and shoulder joints, the smaller articulations of the feet and hands remaining intact.

4. When established, there is a well-marked curve of the spinal column antero-posteriorly, with occasionally a slight lateral list; its contour is almost regular, but slightly accentuated in the cervico-dorsal region, as if the upper part of the column were drawn forwards and downwards by the weight of the head. In some cases the cervical portion of the column undergoes luxation, with a corresponding accentuation of the deformity, and the patient's face may look directly downwards; there may or may not be obliteration of the lumbar curve. In advanced cases the ankylosis is absolute, and the faulty attitude is unaffected by any degree of force, short of fracturing it. In the early stages, however, when the bodies of the vertebræ are undergoing softening, the faulty position allows of rectification, but later on, owing to ossification of the ligaments, such is prohibited.

5. The hip joints may be ankylosed in flexion or extension, and in accordance with this Marie differentiates two types, *type en flexion* and *type en extension*; the shoulders are affected, but to a lesser degree (*Plate XXXV, Figs. A and E*).

6. The thoracic cage is always more or less flattened from before back, sometimes to a considerable extent, and the same holds good of the pelvic basin. Marie comments on the similarity existing between the radiograph of an osteomalacic pelvis and one affected by this disease, and quotes Ascoli to the effect that such confusion has occurred. In addition to the pharyngeal exostoses, similar formations have occasionally been felt in the vicinity of the sacro-iliac synchondroses.

The general appearance of the patient is extremely striking, his fixed attitude being compared by Marie to that met with in sufferers from paralysis agitans. When the cervical portion of the column is affected, the subject is unable to extend his head, peering from beneath his pent brows at any object in front of him, and in order to see anything above the level of his head he is compelled to flex his knees, and throw his whole trunk and head backwards, so as to bring the object in line with his eye.

Owing to the fixation of his hips, the patient's mode of locomotion is extremely peculiar ; thus in attempting to walk he is compelled to take short steps, pushing his feet forwards, jerking one side of his trunk forwards, then the other, or as Teissier expresses it, he progresses in a zig-zag manner, while Marie compares his mode of locomotion to that of a wooden mannikin.

Anyone who has seen such cases will have been struck by the deplorable degree of crippledom to which these unhappy victims are reduced. The sufferer cannot sit on an ordinary chair, but must rest on sloping surfaces. When lying on his back the patient cannot raise his head, and rotation is impossible without movement of the whole trunk *en masse*. The rigidity of the whole spine is so absolute that, in one case of my own, if the head and neck were depressed the lower part of the column became elevated, and *vice versa*. Should the knees be affected as well as hips and spine, the patient's misery is still more aggravated, as it is then impossible for him to raise himself even a few inches to surmount any obstacle, and in order to ascend the stairs he has to be assisted on both sides.

With regard to the spondylosis due to *osteo-arthritis of the spine*, as Goldthwait[5] remarks, the deformities produced may be very various. In the most marked examples of this variety we get a widespread and extreme kyphosis, with projecting head and flattened chest. According to French authors, it is in this hypertrophic type, characterized by voluminous exostotic outgrowths, that we meet with a fixation of the spine in a rigid straight line, with obliteration of the normal curves—the so-called poker back. It is in this osteo-arthritic variety that root-pains due to pressure by exostoses are so common, and the widespread palsies of the limbs referred to in the general description are usually, if not exclusively, confined to this hypertrophic or osteo-arthritic type.

Finally, a condition of spondylitis is met with in *rheumatoid arthritis*, as I[6] have before pointed out (*Plate XXXVI*). Either coincidently with or subsequent to a polyarticular rheumatoid arthritis of the limbs, a condition of more or less marked rigidity of the spine may ensue. In such cases the onset of the rigidity is usually preceded by severe neuralgic pains, traversing usually the cervico-occipital and brachial regions, and I have seen permanent anæsthesia in the limbs follow these pains. The curvature is usually most marked in the cervico-dorsal region, where there is a variable degree of kyphosis, with projection forwards and downwards of the head, and this occasionally to a very marked degree. The chin, besides being depressed, may show a decided tilt to one or other side. The atlanto-occipital as well as the other cervical articulations may be involved, and the power of rotation of head may be almost abolished. The spinal, scapular, and pectoral muscles may show conspicuous atrophy, with marked increase of myotatic irritability, and the wasted and shrunken appearance of the back of the neck contrasts with the often very marked thickening of the nuchal region met with in the osteo-arthritic type. The rigidity may extend to the whole column, likewise the costo-vertebral articulations.

Arising as these cases usually do as part of a generalized polyarthritis, a variable degree of involvement of the joints of the limbs is usually present, the shoulders, hips, and other large joints being usually implicated, while happily in these cases the hands as a rule are not affected.

When, as in a case I saw recently, total rigidity of the spine, with involvement of the hip and shoulder, is present without implication of any other joints, the resemblance to the spondylose rhizomélique type is most striking.

It now remains for us to consider how far these distinctions between the various types hold good, and it must be admitted, without in any way seeking to underestimate the value of the work done by von Bechterew, and Strumpell and Marie, on their respective types, that increasing observations tend rather to show the impossibility of drawing any hard and fast line between their varieties, owing to the existence of intermediate and transitional forms testified to by so many observers.

Thus, for example, von Bechterew insisted that in his variety there was no involvement of the joints of the extremities, but Popoff narrates a case of this type in which, during the development of the spinal rigidity, the patient's thumb was painful and swollen for some weeks; furthermore, the history of the case disclosed the fact that six years previously he had an affection of the wrists which terminated in permanent flexion of those joints, both of which facts, as Zenner points out, indicate an arthritic tendency.

Again, Sachs and Fraenkel[7] relate a case of the von Bechterew type in which the spinal rigidity and root pains developed in the lumbar region of the column, and here again in addition the shoulder joint was implicated during the early stages of the malady. We have, moreover, in this particular instance the unusual advantage of knowing that the underlying pathological changes were very similar to those obtaining in the von Bechterew type, inasmuch as thickening of the meninges was disclosed during the course of a laminectomy undertaken by Dr. Gerster at the Mount Sinai Hospital for the purpose of relieving severe root pains. It is pleasing to relate that as the result of this operation the rigidity markedly lessened, and the knee-jerk, which had been abolished during the course of the malady, reappeared.

Although these observers hesitate as to whether or not this case should be included in the category of the von Bechterew type, it seems to me that it may legitimately be classed as such, inasmuch as von Bechterew himself states that the malady described by him may affect either a portion or the whole of the spinal column. It is obvious from these two examples that the changes in the von Bechterew type are not necessarily confined to the spinal column, but may be associated with disease likewise of the joints of the extremities.

Again, I myself have observed cervico-dorsal kyphosis with rigidity of the spine develop in the course of a generalized rheumatoid arthritis, and attended by severe cervico-occipital and brachial pains, followed by permanent anæsthesia, the area of which corresponded more or less exactly to the eighth cervical territory (Head).

From the clinical point of view we see, therefore, that as far as rigidity of the spine, cervico-dorsal kyphosis, radiating pains, and anæsthesia are concerned, this case resembled the von Bechterew type, differing only in the widespread involvement of the joints of the extremities.

With regard to the spondylose rhizomélique type, Strumpell claimed that the changes were confined to the spine, hip, and shoulder joints. Teissier,[8] however, in one of his more recent contributions on the subject, states that the knees also are almost always affected, and the temporo-maxillary and sterno-clavicular joints not infrequently, and, in addition, he writes that one commonly meets with cases of this type in which the morbid changes have extended to the smaller articulations of the hands and feet. Valentini[9] has also reported an instance in which the small joints were involved. Moreover, apart from the anatomical distribution of the lesions, an absence of root pains cannot be claimed as pathognomonic of this affection as opposed to the von Bechterew type, although, as will be seen in the historical section, Strumpell maintained in his original description that the disease arose gradually and painlessly, for on the other hand Marie distinctly states that the ankylosis is preceded by a period in which pains predominate.

Teissier also notes the occurrence of these root pains, not to mention Magnus-Levy,[10] who states that nerve root symptoms are to be met with in both the von Bechterew and Strumpell-Marie types, and that they are not invariably present even in the former variety. Indeed, it would seem that not only may nerve root symptoms be met with in spondylose rhizomélique, but also signs indicative of spinal cord involvement, as in a case of this type reported by Eshner, in which the widespread wasting, heightened reflexes, and physical signs generally led him to suspect not only disease of the vertebræ and appendages, but also of the spinal cord.

The foregoing observations by various authorities seem to conclusively prove that no distinctions can be drawn on the grounds specified between the Strumpell-Marie and von Bechterew types. Indeed, based as they are for the most part on mere superficial differences in the anatomical distribution of the joint lesions, it could hardly be doubted that more extended observations would demonstrate their futility.

It now remains to consider their relationship to spondylitis deformans regarded as a whole, from which condition some few authorities would sharply divide them off as distinct morbid entities. As I have shown, varying degrees of spinal rigidity are to be met with in the subjects of rheumatoid arthritis, and when this is localized to the cervico-dorsal region the deformity and associated root symptoms present a clinical picture which closely resembles that of the von Bechterew type. Similarly, in those rare instances in which the whole spinal column is involved in rheumatoid arthritis, with, as I have seen, implication of the shoulder and hip joints only, we find the clinical aspect of spondylose rhizomélique exactly reproduced.

This clinical similitude on the part of these three types seems to suggest that all distinctions drawn between them are purely artificial. Moreover, this supposition is strengthened by the fact that similar pathological changes are to be found in the vertebræ, these being of a predominantly atrophic character. It would appear, therefore, legitimate both on clinical and pathological grounds to designate them as examples of the rheumatoid or atrophic form of spondylitis, as opposed to the osteo-arthritic or hypertrophic variety. The clinical differentiation of these two main groups we have endeavoured to indicate in the foregoing text, and should further research confirm Marie and Léri's able work in regard to their pathological distinctness, it would show that the same great cleavage into atrophic and hypertrophic divisions obtains, not only in the peripheral form, but also in the central or vertebral variety of arthritis deformans.

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CHAPTER XXII.

DIAGNOSIS.

THE diagnosis of spondylitis deformans in its earlier stages is beset with difficulties, and the disorders liable to be confounded with it will naturally vary with the particular region of the column affected.

The chief sources of error when the disease attacks the cervical vertebræ are tubercular caries and meningitis of this region. Both Gowers[1] and Ruhrah have emphasized the possibility of confusion with meningitis. Similarly, when the dorsal segments are affected, care must be taken to exclude not only Pott's disease, but spinal rigidity secondary to thoracic aneurysm, and, according to Gowers, involvement of the vertebral column by malignant disease must not be forgotten.

The fact that pain and spinal rigidity are common to all the above diseases, and in their earlier stages may indeed be the only clinical features indicative of disease of the column, fully justifies the caution inculcated by these two authorities.

When the disease affects the lumbar segments of the spine it is important to be alive to the many causes of spinal rigidity due to reflex spasm, such as movable kidney, renal calculus, and other disorders. In addition, the subacute or more chronic forms of osteomyelitis must be taken into consideration, inasmuch as Labeyrie[2] has noted that infections of the medulla of the vertebræ occur most frequently in this region of the spine. Moreover, when the lumbar column is the seat of spondylitis, more particularly of the hypertrophic or osteo-arthritic variety, severe sciatic pain may be the only symptom, the patient himself being often unconscious of any restrictions of the movements of the spine ; thus it frequently happens that the true nature of the malady is overlooked, and the condition deemed to be one of primary sciatica. In conclusion, stiffness of any section of the vertebral column due to chronic muscular rheumatism must be excluded, likewise the condition known as hysterical spine.

The above brief summary of the pitfalls in diagnosis which may entrap the unwary sufficiently demonstrates the necessity, if we would escape error, of thorough examination in all cases of rigidity of the spine.

Going more into detail in connection with this question of spinal immobility, the first point to ascertain is whether the stiffness of the column is organic and permanent, or, on the other hand, functional and temporary.

In attempting to solve this problem, one cannot do better than adopt Sir William Bennett's[3] method of examination for differentiating these two varieties. His method of procedure is as follows :—

"The patient, stripped to the hips, standing with the back to the observer, should bend forward in the usual way, the lower limbs being kept extended at the knee, the hand of the observer at the same time resting on the rigid portion of the spine. The bending forward of the trunk having been carried out as far as possible, the patient should not immediately begin to resume the upright position, as is usually done in spinal cases, but should maintain the bent position for a considerable period, at all events for two or three minutes. It will then be found that if the rigidity be due to muscular action, a slight quivering of the muscles lying under the hand of the investigator will occur ; this quiver will, if the bent position be maintained, be replaced by a series of jerks ('stammering'), until in some cases the flexibility of the vertebral column can be freely established."

This authority further states that "the occurrence of this symptom of stammering may be held to be absolute proof of the absence of organic disease of the vertebral column."

In this manner we may exclude rigidity of the spine due to hysteria, chronic muscular rheumatism, and reflex spasm, such as may be associated with movable kidney, etc.

Oppenheim[4] emphasizes the fact that the clinical picture presented by chronic muscular rheumatism may resemble in every particular that presented by spondylitis deformans, and in order to clear up the diagnosis he advises the administration of an anæsthetic, when the rigidity, if due to the former, disappears, but if due to the latter it persists during narcosis, and therefore, when pain precludes the possibility of the application of the "stammering" test, resort should be had to an anæsthetic.

Having now established that the rigidity is permanent in character, it will be necessary to decide as to the nature of the underlying morbid process, and as far as possible to differentiate the various affections from spondylitis deformans, the general characteristics of which are as follows:—

1. It is a disease of youth and early middle life, most frequently met with in men, and but rarely in women.

2. It gives rise to pain and stiffness of the spine of a progressive character, leading to fixation of large sections, or the whole, of the spinal column, and the production of more or less deformity, usually in the direction of kyphotic curvature, whose contour is generally smooth and rarely marked by prominence of individual spines. It is often accompanied by ankylosis of costo-vertebral articulations, leading to immobility of the chest walls and abdominal breathing.

3. Nervous symptoms, variable in character, intensity, and distribution, accompany the malady, such as root pains, atrophy of muscles, disorders of sensation and motion.

4. Palpable exostoses, either pharyngeal in site or scattered over the posterior surface of the spine and sacrum.

5. Presence of, or history of, previous arthritic lesions elsewhere. To these may be added the anatomical changes revealed by skiagraphy, more especially those due to exostotic outgrowths.

DIFFERENTIAL DIAGNOSIS.

Tubercular Caries.—More common in children and adolescents; no special predilection for the male sex. Disease most commonly localized to the lower dorsal segments (Bradford and Lovett).[5]

Characteristic angular deformity of the affected portion of the spine.

Lateral deviation of the spine, due to the marked muscular spasm which is so constant a feature in the disease.

Paralysis of limbs, either of central or peripheral origin: the central or paraplegic form is much more commonly met with in caries, and occurs very rarely in spondylitis, in which, if any paralysis appears, it will usually be found to be peripheral in type (Goldthwait). Oppenheim also notes that this peripheral form predominates in spondylitis, and that compression of the cord itself is but rarely met with.

Again, according to Goldthwait,[6] if in any given case of spinal rigidity these two varieties of paralysis are present, such as peripheral palsy in the upper limbs with a paraplegia of the lower limbs, such commingling of the two types points more to a condition of osteo-arthritis of spine with exostoses causing pressure symptoms than to one of tubercular disease.

Presence of tubercular lesions elsewhere will be of great importance in affording a clue to the true nature of the underlying trouble. In advanced cases, skiagraphy will demonstrate the presence of extensive destruction of bone.

Malignant Disease of Spine.—Occurs usually in later life, almost invariably in sequence to malignant disease of other organs.

Extreme severity of pains, rapid wasting, and general cachexia will afford assistance in determining the gravity of the affection.

Typhoid Spondylitis.—History of attack of enteric fever. Intense local pain over the lumbar spines, with radiating neuralgic pains of extraordinary severity. Some rise of temperature and swelling of soft parts over affected region, together with, according to Quincke, rapid disappearance of the spinal symptoms. McCrae[7] has detected by skiagraphy new bony formation in one of these cases.

Traumatic Myositis Ossificans.—History of injury. Presence of bony formations in muscles in the neighbourhood of immobilized portion of spine, as revealed by palpation and skiagraphy.

Occasionally, as in a case of my own resulting from a hunting accident, severe radiating pains may ensue for a prolonged time, and in this particular instance led to crippledom extending over some years, and a condition of tabes dorsalis was suspected. Examination revealed that the lumbar spine was perfectly rigid, and the quadrati lumborum were largely replaced by masses of bone.

Osteitis Deformans.—Generalized osseous hypertrophy, including the skull: increase in size of long bones, chiefly diaphyses, with bowing of the legs: no involvement of the joints as a rule.

Cervical Pachymeningitis.—If it be remembered that the existence of meningitis has been established post mortem in cases of spondylitis, it is obvious that from time to time symptoms of meningeal involvement of the roots will be met with. In cases presenting these marked nervous phenomena the presence of thickening of the soft tissues in the nuchal region, the detection of enlargement of bones by palpation or skiagraphy, together with presence or history of arthritic lesions elsewhere, will be the points on which we should chiefly rely in making our diagnosis from chronic meningitis unassociated with, or, more accurately, independent of, bone disease.

Syringomyelia.—Ruhrah, in discussing the diagnosis of spondylitis, says: "Indeed there is considerable danger of mistaking some cases of syringomyelia for spondylitis if the former disease is not borne in mind." It can hardly arise except in those cases in which there is present scoliosis or kyphoscoliosis, and the diagnosis can only be arrived at by a detailed examination of the whole nervous system, especially as to the presence or absence of dissociated anæsthesia, etc.

Sciatica.—We have before pointed out that sciatic pain may mask an insidious osteo-arthritis of the lumbar spines, and we would here lay stress on the importance of fixing the pelvis in such cases, in order to ascertain the mobility of this portion of the spine in all directions, with X-ray examination if any rigidity is detected. It is at least worthy of mention that a secondary scoliosis of the spine may develop during the course of sciatica.

In conclusion, a few words may be said on the *Tuberculin Test* which McCrae employed in his series of cases, but with a negative result. He nevertheless considers it a great help in the matter of diagnosis. Poncet and Mailland have recorded a case of spondylose rhizomélique in which a very positive reaction was obtained by P. Courmont.[8] Oppenheim, however, discussing the employment of this test in tubercular spondylitis, says, "Never inject tuberculin to confirm a diagnosis," but he realizes the advantages of skiagraphy.

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CHAPTER XXIII.

TREATMENT.

IN this terrible variety, as in all other forms of arthritis deformans, early recognition of the true nature of the affection is essential, especially if any attempts to limit the resulting deformity are to be successful.

In the first instance all possible sources of local infection or absorption must be excluded. In this connection it may be mentioned that Baer and Rosenheim have reported a case of spondylitis in which relief followed the detection by Rosenheim of an infective focus in the ethmoidal cells. Bloodgood, to whom I am indebted for the above reference, narrates an instance of pain and stiffness in the upper cervical vertebræ, in which antiseptic treatment of a decayed tooth was followed by disappearance of the symptoms. The possibility of a gonorrhœal infection must be borne in mind, and if detected, its effectual treatment is the first step to be undertaken. The same general measures must be taken to promote a good state of health, such as abundance of nourishing food and plenty of fresh air and sunshine. Medicinally we have to rely upon prolonged administration of arsenic in combination with syrup of iodide of iron, with or without the addition of cod-liver oil. In the acute stage of the malady fixation of the spine is called for. In the milder cases this may be attained, as Goldthwait[1] suggests, by the wearing of webbing or canvas belts or corsets. In the more severe cases a plaster jacket may be fitted; afterwards a leather one should be substituted. The jackets, according to Lovett and Bradford,[2] should be applied without suspension. No force should be used, but the patients should stand as erect as possible while the casing is being fitted. By these mechanical means the rest necessary for the spine is obtained, and the jacket should be continued until the acuteness of the process has subsided, when a lighter form of corset should be adopted.

Fixation is also of value in that it lessens pain, and moreover, inasmuch as the curvature may be accentuated by muscular spasm, the support often tends to diminish the degree of this, and thus corrects a certain measure of the deformity. Pierre Marie and Léri,[3] when dealing with the treatment of spondylose rhizomélique, lay stress on the importance of taking the weight of the head off the spine. They quote a case of this affection under Baumler's care in which the patient was placed in the recumbent position with the neck resting on a transverse bar, but the head unsupported and allowed to hang backwards. As a result of the influence of gravity the forward stoop of the head and neck was in this way redressed.

Marie considers that this method should be more extensively employed, but it should be borne in mind that it is only in the early stages that it is of any value. At this period of the disease the bodies of the vertebræ are undergoing rarefaction and softening, and as a result of the action of gravity a gradual falling forward of the head and neck takes place. This deformity is at first capable of being corrected, but later on, owing to the secondary ossification of the ligaments, it becomes impossible.

Marie also issues a warning against excision of the hip in these cases, inasmuch as after the resection of the articular surfaces ankylosis may result subsequently owing to ossification of the capsular and ligamentary structures. In cases of the osteo-arthritic variety of the malady attended by severe radiating pains, counter-irritation by blisters or the actual cautery should first be tried perseveringly. If these should prove ineffectual and X-ray examination reveal the presence of bony outgrowths encroaching on the neighbourhood of the nerve roots, the question of an operation to remove the osseous nodes causing the pressure symptoms will arise. In all forms, when the acute stage is past, massage of the atrophied muscles should be practised, and may advantageously be combined with thermal treatment in the form of Aix massage. No attempt, however, should be made by forcible manipulation to correct deformity or to obtain more freedom of movement in the spinal articulations.

Hot air and radiant baths may also be used at this stage of the disease.

In conclusion, the results of any form of treatment of the disease when advanced are so very discouraging that the importance of early recognition, with timely resort to the orthopædic measures above alluded to, cannot be too strongly emphasized, as in this way only can some of the more distressing features of the affection be minimized or possibly prevented.

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[3] MARIE et LÉRI. La Spondylose Rhizoméliqu . "Nouvelle Iconographie de la Salpêtrière," 1906, No. i., p. 36.

SECTION V.

ARTHRITIS DEFORMANS IN CHILDREN.

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CHAPTER XXIV.

ARTHRITIS DEFORMANS IN CHILDREN.

HISTORICAL.

ACCORDING to Diamantberger,[1] it is to Cornil (1864) that we are indebted for the first recorded instance of rheumatoid arthritis in early life, that physician having noted its onset in a girl at twelve years of age. In the same year Laborde[2] exhibited at one of the French Societies a child in whom the disease began at the age of four. Adams[3] in 1857 stated that he had seen several instances under twenty years of age, but he gives no further details beyond saying that the disease might be widely generalized or localized. Trousseau[4] observed its incidence at the age of fifteen; Charcot[5] relates two instances at ten years of age, and Picot[6] one at seven. In 1876 Stoicesco[7] contributed a short study on the subject, citing three cases of his own, two at four and one at five years of age. Lewis Smith[8], of New York, in the same year records its occurrence at four years of age. It is, however, to the efforts of Moncorvo[9] that we are indebted for the first comprehensive monograph on the subject, which appeared in 1879. Subsequently, Lacaze Dori,[10] in 1882, contributed a thesis on the subject, likewise Pelissie,[11] in 1889, and Henoch,[12] in 1883, recorded two instances in a boy and girl aged thirteen and fourteen respectively. In 1891 appeared a very elaborate treatise on the subject by Diamantberger, who laid stress on the fact that all forms of arthritis deformans met with in adults are to be seen in early life. The comparative rarity of the affection is shown by the fact that Koplik,[13] in 1895, could only glean from the literature eighteen cases of rheumatoid disease in children.

In 1897 appeared Still's[14] important communication on the subject, which contains the first attempt at cleavage, and in which he details the characteristics of the variety now associated with his name. This brief sketch of the historical aspect of the disease is, however, very imperfect, but it serves to demonstrate the paucity of the literature on the subject as compared with the overwhelming amount written upon the disease as met with in adults.

It would appear from the writings of various authors that at least four varieties of disease are to be met with in this category: (1) A condition clinically comparable in all its course to rheumatoid arthritis as seen in adults. (2) Cases much more rare which correspond to the osteo-arthritis, both localized and generalized, of adults. (3) "Still's disease," defined by its author as a "chronic progressive enlargement of the joints associated with enlargement of the lymphatic glands and spleen." (4) The cases described by Jaccoud as *rhumatisme fibreux*, in which the joints show peri-articular thickening of a fibrous nature, without any evidence of bony changes.

RHEUMATOID ARTHRITIS.

Little or nothing is known as to the etiology of this condition as it occurs in children. Diamantberger attached considerable importance to environmental circumstances, such as damp, cold, poverty, insufficient food, and unhygienic conditions generally, but Still, on the other hand, noticed a conspicuous absence of such depressing conditions in some of his cases. The former also considered that the condition developed more often in the children of diabetic, rheumatic, and gouty subjects. In addition, he noted that in two of his examples the parents were the subjects of Graves's disease, while in another instance one of the parents of the child was insane.

In all cases of my own, however, I have been unable to find any trace of an hereditary predisposition to arthritic diseases, but I may mention that in one instance the mother of the patient was insane. In seven cases that came under my observation, two gave a history of measles, one of whom was believed to have had influenza in addition; two were said to have had "rheumatic fever," and had suffered with sore throats, while in the remaining two there was no history of any previous illness. Judging from the reported examples, as far as I have been able to ascertain, the disease is almost unknown before the age of two years, though Still records the incidence in a child twenty months old. Instances of its occurrence at the age of two have been observed by Lewis Smith, Moncorvo, and others. The youngest child I have seen affected by it was seven years old. As regards sex, all the cases in my short series were females. Still mentions that in his six examples four were girls and two boys. Diamantberger, out of thirty-five cases collected from the literature, including his own, noted that twenty-four occurred in girls, and eleven in boys.

From the above observations it would appear that the affection in children, as in adults, occurs more commonly in the female sex.

MORBID ANATOMY.

The rarity with which opportunities occur for post-mortem examination of examples of early rheumatoid arthritis in children justifies one in quoting the following reports at some length.

Delcourt,[15] in his treatise on rheumatoid arthritis in children, records the case of a girl, four years old, the subject of a chronic polyarthritis.

The disease began with swelling of the right ankle joint, and, subsequently, invaded both knees, the hands, and cervical articulations.

Eighteen months after the onset the child succumbed to acute osteomyelitis, the sequel of an accident.

The autopsy showed the presence of pulmonary congestion, also pericardial adhesions. The endocardium was, nevertheless, normal; but the liver was the seat of fatty infiltration.

Joint Changes.—On section, the subcutaneous tissues of the right ankle joint were found to be markedly œdematous, discharging abundantly serous fluid. The tendons and their sheaths were normal. The capsules of the tarsal and metatarsal joints were thickened, but contained no excess of fluid. The cartilages covering the articular surfaces of the calcaneum and the navicular and cuneiform bones were the seat of erosions chiefly central in site.

The Left Knee Joint.—No adhesions were present, the synovial membrane being smooth. No intra-articular effusion, but slight in which he details the characteristics of the variety now associated with his name. This brief sketch of the historical aspect of the disease is, however, very imperfect, but it serves to demonstrate the paucity of the literature on the subject as compared with the overwhelming amount written upon the disease as met with in adults.

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The Left Knee Joint.—No adhesions were present, the synovial membrane being smooth. No intra-articular effusion, but slight erosion of the epiphyseal cartilages of the femur and tibia was present, the patella, however, remaining intact.

Right Knee Joint.—The articular cartilage showed slight erosion on its outer side.

Right Index and Middle Fingers.—The mid-phalangeal joints were much enlarged, owing to thickening of the peri-articular tissues. In the middle finger osteomyelitis was present, but localized to the lower end of the first phalanx and upper end of the second, the cartilages remaining intact. In the index finger, however, the changes had gone on to destruction of the cartilage covering the upper end of the second phalanx. Sections of the peri-articular structures revealed the presence of fibrous thickening. In and between the connective tissue bundles were scattered spindle-shaped cells with large nuclei.

Nowhere was there anything of the nature of granulation-tissue or tubercular nodules to be seen. On the contrary, the tissue was purely fibrous, sparsely provided with blood-vessels, the walls of which had undergone fibrous thickening.

Tubercle bacilli were absent, but chains of streptococci were found in the bony marrow.

In addition, Johannessen[16] has reported in great detail two cases of the affection in children. The first relates to a girl, seven years old, who, after an illness of three years' duration, fell a victim to tuberculosis, complicated by hæmoptysis.

Briefly described, the following changes were found in the right hip and knee joints when laid open.

Macroscopically, the capsule showed connective-tissue thickening, while the cartilage, to a large extent, had lost its normal sheen, was rough, uneven, and here and there completely atrophied, being replaced by connective tissue.

Tubercular nodules were absent from the synovial membrane.

Microscopically examined, sections of the capsule of the hip joint revealed the presence of connective tissue with scanty formation of cells, these latter individually, somewhat large in size, but not comparable to giant-cells.

Sections were made through the affected portions of the cartilage, which in one area was yellowish in colour and semi-transparent.

Microscopically, it was seen that the outermost layer of hyaline cartilage had undergone gradual transformation into fibrous tissue, the latter extending at the expense of the former.

This newly-formed tissue, at first poor in blood-vessels, subsequently becomes more vascular. This metamorphosis of cartilaginous into fibrous tissue was in places abrupt, with a sharply-defined limit.

In the deeper layers of the cartilage the cells were of irregular character and arrangement, while some were seen to have undergone degenerative changes.

Ultimately, the layer of cartilage cells is replaced by connective tissue overlying the bony trabeculæ, and enclosing here and there among its fibres effete cartilage-cells.

Johannessen's second case was a boy, six years of age, whose illness had existed for about a year. In the first eight weeks of the malady pyrexia of high degree prevailed, with marked perspirations; but, in the later months the condition was stated to be afebrile.

The disease pursued a progressive course, the patient becoming very anæmic; and, eventually, death occurred.

At the necropsy all the articulations in the right foot were opened; and the morbid changes throughout were similar in character. The synovial capsule of the joints, as well as the greater part of the ligaments, were thickened, owing to the presence of a gelatinous, soft connective tissue, with numerous projections up to the size of a bean.

In sections this membrane was seen to be made up of fine connective tissue with intervening loculi, and abundantly supplied with blood-vessels.

Section of the astragalus, os calcis, and cuboid showed these bones to have undergone extensive osteoporosis. The bony trabeculæ were excessively fine, enclosing large cavities filled with abundant red marrow.

Right Knee Joint.—The peri-articular and capsular structures with the crucial ligaments were the seat of gelatinous œdema. The cartilage was intact, retaining its smoothness and polish.

The right hip joint exhibited swelling of the capsule and ligaments; but no other alteration. Similarly, the finger joints of the right hand were marked by slight swelling of the softer tissues, the articular ends being normal.

The same description applies to the right wrist, shoulder, and elbow-joints.

Speaking generally, a considerable amount of fluid was present in the joints. The effusion was viscous, yellowish, with some fibrin-flocculi. Microscopically, it contained large, round cells, but no bacteria ; and, in the opinion of Johannessen, it could not, on microscopic evidence, be characterized as purulent or puriform. While, in addition, both sternoclavicular joints were the seat of an effusion, the cartilage on the right clavicle showed erosions of the size of a hemp-seed.

Cultures were taken from the joint-effusions in tubes of agar and of bouillon. One tube of agar showed the presence of bacilli, stained by Gram's method ; likewise cocci, which responded to the same reagent.

Spitzy[17] records the case of a young girl, aged 8, in which the joint-condition had pursued an acute course for nearly nine months. Arthrotomy of the right knee joint was then performed, which provided an opportunity for observing the changes in the articular cavity. The synovial membrane was hyperæmic, and the seat of slight villous overgrowth, but not comparable in degree, according to Spitzy, with that obtaining in Schüller's "arthritis villosa chronica."

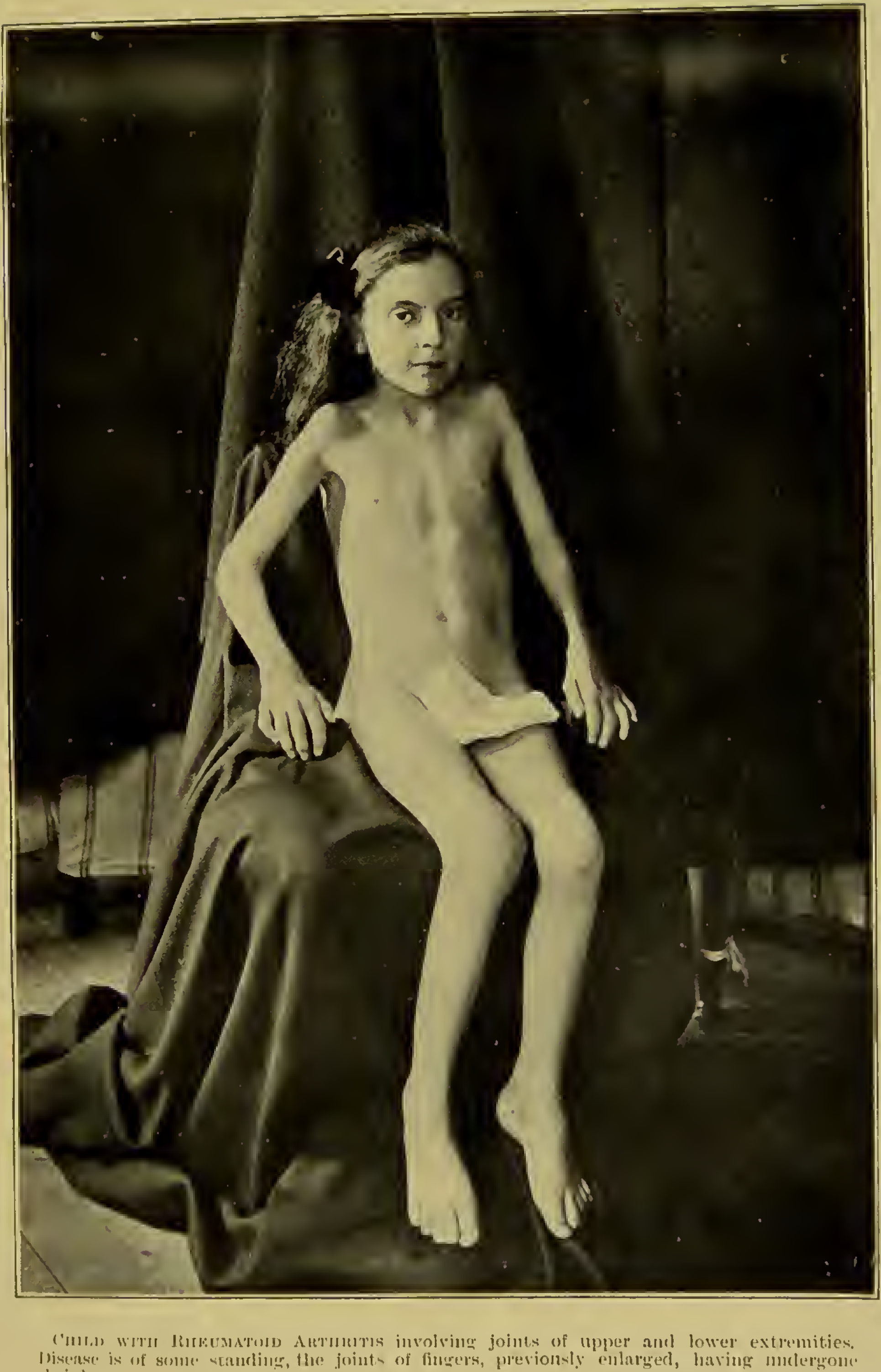
The joint-surfaces were throughout smooth and covered with intact cartilage.

Microscopic examination of the synovial membrane revealed an extraordinary number of nests of round cells throughout the whole extent and thickness of the membrane. In the superficial layers beneath the endothelial lining the cell-nests were most abundant, while in the deeper layers fat-cells and connective tissue predominated. Elastic fibres were very meagrely represented.

In reviewing these records of autopsies, it will be noted that the most salient pathological fact is the intact condition of the inter-articular cartilages in the early stages of the affection.

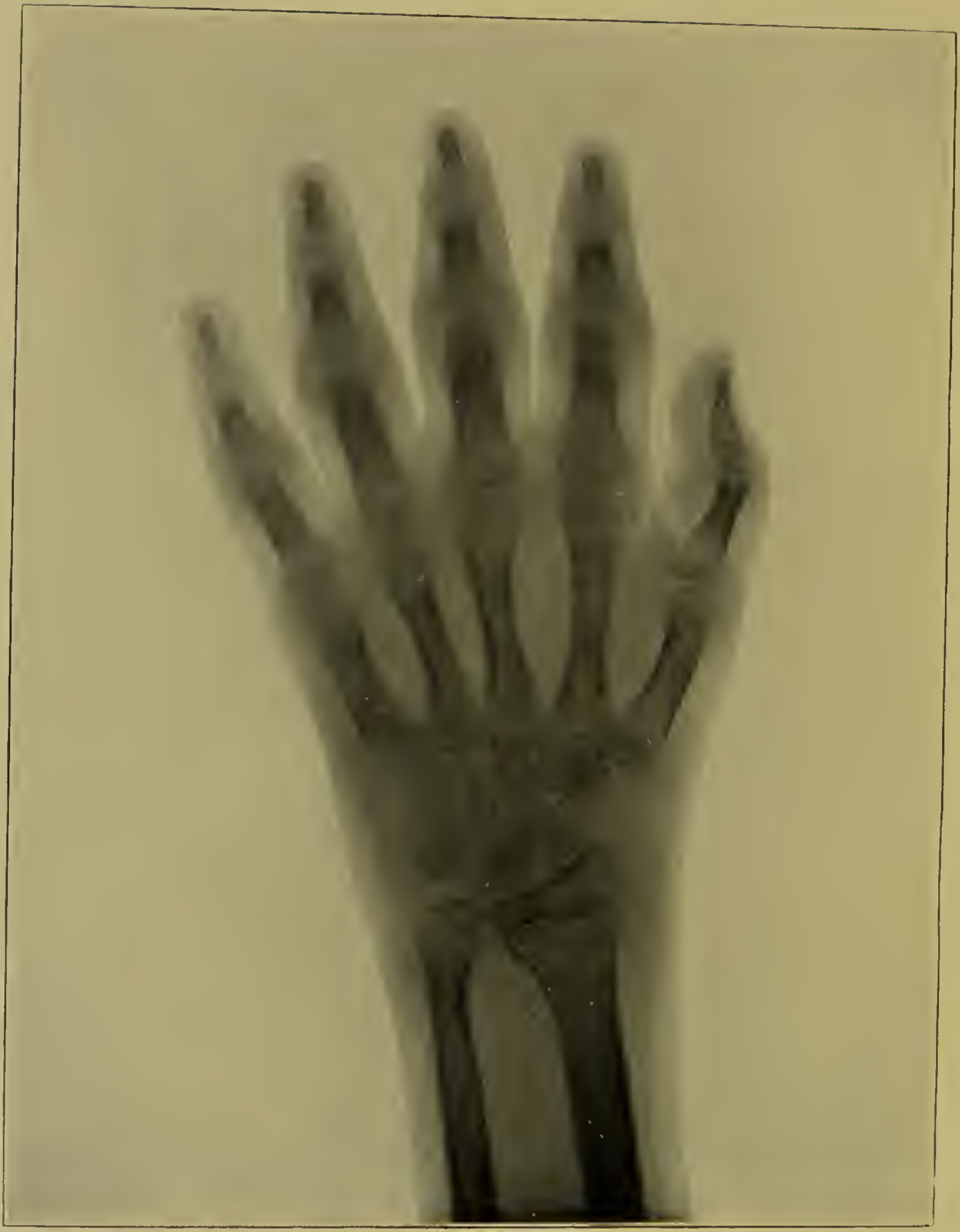
It seems fairly certain that at first they remain wholly unaffected ; but in joints the seat of more advanced disease they undergo erosion. The changes, therefore, are strictly comparable to those observed in the rheumatoid arthritis of adults, when examined at an early stage of the affection.

PLATE XXXVII.



CHILD WITH RHEUMATOID ARTHRITIS involving joints of upper and lower extremities. Disease is of some standing, the joints of fingers, previously enlarged, having undergone shrinkage.

PLATE XXXVIII.



SKIAGRAPH OF HAND OF CHILD, aged 11 years, the subject of acute rheumatoid arthritis: showing transparency of ends of bones, and no bony enlargement.

The primary change takes place in the synovial membrane, and the cartilage is only secondarily involved; moreover, the morbid process in the latter is, as a rule, purely regressive in character, and unattended by new bony formations, contrasting in this respect with osteo-arthritis.

The streptococci found in the bone-marrow in Delcourt's case may, I think, be justifiably attributed to a secondary infection, and the same is probably true of Johannessen's second example.

CLINICAL ACCOUNT.

In all of my patients the disease apparently arose insidiously and without ostensible cause, which accords with the experience of Diamantberger as to the usual mode of onset, though he has noted in a few instances a more or less acute initial stage. In my own series of examples I was unable to obtain a history of any premonitory symptoms, but Diamantberger notes that the disease in children is often preceded by very painful muscular spasms as in adults. The condition when mature is an exact reproduction in miniature of what we observe in adults affected with the malady, but the articular lesions in their spread do not conform so closely to the centripetal path of invasion seen in adults, the disease in quite a few instances beginning in the larger joints. The spinal articulations also may be involved. In two of my cases the movements of the cervical portion of the column were almost abolished, in one the head being bent permanently forward until the chin nearly touched the sternum. In the other, while flexion and extension were possible, the power of rotation was apparently lost. The same fusiform or spindle-shaped swelling is seen in the mid-phalangeal and other joints, and no bony enlargements can be felt. The results of skiagraphy, moreover, seem to bear out this absence of bony overgrowths, at any rate in the early stages. The temporo-maxillary articulation may be involved.

The same muscular atrophy, rapid and widespread as in adults, develops, and it displays the same selective affinity for attacking the interossei and extensor groups at an early stage of the disease. In two of my cases there was present a notable degree of atrophy of the spinati muscles and the triceps. Similarly, neuralgic pains, either confined to the peripheral portion of the extremities or shooting along the length of the limb, may be complained of, also cervico-occipital neuralgia in cases with spinal involvement. I have met with one instance in which pigmentation was present, and another in which there was great overgrowth of hair on the backs of the forearms. Sweating of the extremities was a feature in one, while the hands of these patients are usually cold, but I have met with no cases presenting such marked vasomotor phenomena as may be seen in adults.

There was no splenic or glandular enlargement in any of my cases. Similar malpositions to those found in adults may develop, such as the characteristic ulnar deviation of the hand and fingers. In one of my examples this condition was associated with hyperextension of the wrist, while the elbow was semiflexed and held midway between pronation and supination.

In another instance both wrists were flexed almost to a right angle with the forearm, with some radial declination at the wrist. Diamantberger notes the occurrence in his cases of paroxysmal palpitation without any valvular mischief, which he compares to the tachycardia of Graves's disease. In one of his patients double exophthalmos was present, and enlargement of the thyroid gland is, according to him, not so very infrequent. In another of his examples the patient's parents, also his aunts and cousins, were the subjects of goitre with or without exophthalmos, these manifestations appearing to alternate with arthritic troubles of the most varied character. These and other observations of a similar nature led him to think that there might exist a morbid affinity between rheumatoid disease in children and exophthalmic goitre. Trophic changes in the skin are also to be met with in the later stages. In one of my cases such were present. According to Diamantberger, the alterations in the ends of the bones sometimes lead to arrest of growth in the skeleton, and he notes that an inequality in the length of the femora and tibiæ may result. He also lays considerable stress on a lack of development in the lower jaw which he observed in three of his cases.

STILL'S DISEASE.

This special variety of chronic arthritis in children was first described in 1896 by Still, who considers it to be a peculiar disease occurring in children which begins usually before the period of the second dentition, and is characterized clinically by elastic fusiform enlargement of the joints without bony change, and also by enlargement of the glands and spleen. His clinical account of the affection was based on a series of twelve cases, three of which came to autopsy. Of the examples, seven were girls, five were boys. The earliest age at which onset of the disease took place was at fifteen months. Ten out of twelve began before the age of six years, and eight of these arose during the first three years of life. The youngest case that I have seen was a girl of seven, and one boy was eight years old. In one of my examples there was a previous history of abscesses, one situated in the scalp and one in the popliteal region. McCrae, in his series, records two cases, one a male aged four, and the other a female aged five. Both had rickets, but nothing suggestive of congenital syphilis.

Its causation appears to be obscure. Still remarks that heredity does not appear to play any part in its etiology, neither can it be ascribed apparently to unhygienic conditions. But little if anything has been added to Still's original description, the main features of which are as follows: The onset of the disease is usually insidious, but it may be acute, with chills and pyrexia. It begins with stiffness in one or more joints, gradual enlargement of which ensues. The wrists, knees, and cervical spine are usually first affected, subsequently the fingers, toes, and other joints. The sterno-clavicular and temporo-maxillary articulations are in rare instances implicated. The character of the articular swellings suggests thickening of the peri-articular tissues rather than bony enlargement, being smooth and fusiform without any bony irregularity. Creaking on passive movement may be elicited, but never bony grating. The disease is almost always symmetrical. Neither suppuration nor bony ankylosis occurs.

With the progression of the disease, considerable wasting appears, and contractures may develop, but the electrical reactions are unaltered. Enlargement of the lymphatic glands is always present, in the first instance chiefly of those in relation to affected joints, but it may become generalized later. The glands are discrete and hard, but not tender, and show no tendency to break down. Enlargement of the spleen was present in nine out of twelve cases, its enlargement varying with that of the glands and the acuteness of the joint-condition. The temperature in some cases may be almost continuous, rising to 100° or 101° F., but it may take the form of short periods of pyrexia alternating with afebrile periods.

Anæmia of moderate grade is generally present. The heart shows no tendency to valvular disease, but hæmic bruits may be heard.

In two cases of the series there were physical signs pointing to adherent pericardium, and in three pleural adhesions were found at autopsy. Profuse sweating often occurs. A curious occasional symptom is slight prominence of the eyes, unassociated with any thyroid enlargement. Marked arrest of bodily development is often a feature when the disease has arisen before the second dentition. The course is usually slow.

Improvement of a temporary nature is common, but the author met with no instance in which recovery took place. The disease is not in itself fatal, but hopeless crippledom may develop in the course of months or years. In three of the cases definite improvement in the joint condition occurred after the incidence of measles, scarlet fever, and catarrhal jaundice. Whitman[18] describes two cases, one of which was fatal, and the other recovered. In the former, fusiform enlargement of the joints, especially wrists and knees, together with implication of the small joints of the fingers and toes, was present, also extreme muscular atrophy. The joints chiefly affected were opened at different times and swabbed with pure carbolic acid, and subsequently washed out with salt solution. This procedure lessened the discomfort, but did not influence the course of the disease, which progressed to a fatal issue. It was noticed before death that the skin became excessively thin and transparent. Besides the splenic and glandular enlargement, there was present great enlargement of the liver. No micro-organisms were found. The other case was similar: the left knee was operated on, and eventually recovery took place under a course of electric-light baths. These examples differ, as the author points out, from those previously described, in the following respects: great enlargement of the liver in both, non-involvement of the spine, and complete recovery of one.

It is encouraging to note that one of McCrae's[19] cases recovered completely, the enlarged spleen and glands diminishing with the improvement in the general health and articular condition.

According to Morse,[20] of Boston, there is generally a diminution in the number of red corpuscles, often associated with a disproportionate diminution in the percentage of hæmoglobin.

There is also a leucocytosis, the increase taking place mainly in the polynuclear leucocytes. X-ray photographs of typical cases show no bony changes. The experience of subsequent observers does not uphold Still's statement as to there being no tendency to valvular disease of the heart. Parkes Weber,[21] for example, records a case showing this complication, while Poynton[22], in two of his cases, ascertained the existence of valvular disease, which in one was very severe. Again, in regard to splenic and lymphatic enlargement, it would appear that the former does not occur with such frequency as Still's original description would lead one to expect. Thus, in two cases of my own, splenic enlargement was not present, and in Garrod's experience it is by no means a constant feature. Poynton also states that in cases showing glandular enlargement this is not necessarily always associated with a similar enlargement of the spleen, while Parkes Weber indeed doubts whether it is essential for the diagnosis of this condition that glandular and splenic enlargement should be present at all. It is encouraging to note that the prognosis of these cases is not so gloomy, for, as we have seen, both McCrae and Whitman report one case each in which recovery took place. Poynton and Weber state that the disease varies in severity in individual instances, and that some slowly improve.

MORBID ANATOMY.

Our knowledge of the morbid anatomy is derived from the description by Still of the pathological changes observed by him in the three cases which came to autopsy as the result of intercurrent complications. In two cases of fourteen months' duration the joints, beyond being markedly enlarged, showed thickening of the capsule, increased vascularity of the synovial membrane and ligaments, with slight increase of synovial fluid. In a case of three years' standing there was present increased vascularity and thickening of the synovial membrane, thickened processes of which fitted closely into pits in the peripheral portion of the cartilage, giving in some parts a worm-eaten appearance. The rest of the cartilage was healthy, and the changes in it were considered to be secondary to changes in the synovial membrane. Some fibrous adhesions in the joint were present. Fibrillation of cartilage, osteophytic outgrowths, and eburnation of bone were absent. The glands and spleen, beyond being enlarged, appeared on section to be normal, save a few ecchymoses which were observed in the substance of the former.

The only other post-mortem examination I can find was one performed on the fatal case of Whitman's above alluded to. In this instance there were present amyloid degeneration of the enlarged liver, enlarged spleen, and mesenteric glands. The affected joints contained much mucoid granulation tissue, which had in places produced worm-eaten erosions of the cartilages. The cortical substance of the bones, it is interesting to record, was exceedingly thin, as were also the cartilages. No micro-organisms were found, and the author believed the disease began in the synovial membrane. Microscopic examination of the spleen, according to Morse, has shown hyperplasia of the lymphatic tissue of the Malpighian corpuscles. Excised glands also showed simple hyperplasia.

Edsall and Lavenson,[23] in an investigation conducted in order to ascertain the accuracy or not of Poncet's suggestion that some of the joint affections spoken of as chronic rheumatism, rheumatoid arthritis, and arthritis deformans, might be tubercular in nature, employed tuberculin injections in eighteen cases of this type, including one of Still's variety. In this last instance both splenic and glandular enlargement were present, as well as chronic polyarthritis, and extreme local reaction in the joints of this case followed the injections. These authorities came to the conclusion that many of the cases of Still's disease were due to a modified form of tuberculosis, one of less virulence than the ordinary joint or bone tuberculosis. It may be stated that in the case above alluded to the injections of tuberculin were repeated on several occasions, and invariably intense pain and tenderness in the joints resulted. No organisms were seen in sections of lymphatic glands removed from the axilla in this example, but organisms indistinguishable from tubercle bacilli were detected in an emulsion prepared from the glands; but inasmuch as the injection of portions of the emulsion produced no tubercular foci, the organisms were deemed to be either dead or much attenuated in virulence. In addition, the authors institute a comparison between the condition and Sternberg's type of chronic lymphatic tuberculosis. Weber also considers it not at all unlikely that some cases of this variety are of tubercular nature, the articular lesions being due to tubercular toxins circulating in the blood rather than to the fixation of tubercle bacilli in the synovial membrane. This observer's second case gave a positive reaction to tuberculin. Mouriquand[24] also reports a case which he considers to be of this nature. While Edsall's results are very suggestive, it must be borne in mind that the proof of the tubercular nature of his case is not conclusive. Similarly in Weber's case, while the positive reaction to tuberculin indicates the presence of a tubercular focus somewhere in the body, it does not inevitably follow that the arthritis in this case was of the same nature. Some of Poynton's examples of this variety appeared to him to be of the nature of a rheumatic arthritis aggravated by residence in damp surroundings, but he is inclined to think that in some cases a tubercular origin is possible.

While many of the general features of the disease suggest strongly an infective origin, the results hitherto recorded seem to indicate that the condition may be due to a variety of infections, of which tubercle is probably one.

Before concluding this section, it is necessary to refer to the vexed question as to whether Still's disease is to be regarded as a form of rheumatoid arthritis in children or as a disease quite distinct from it. Still differentiated the condition from rheumatoid arthritis by the absence of glandular and splenic enlargement in the latter, and by the fact that rheumatoid arthritis is characterized by bony thickening and lipping about the joints, and in some instances by the presence of bony grating. It is not, however, obvious how the differentiation can be achieved on this basis, for the following reasons :—

Rheumatoid arthritis, at any rate in its early stages, is not accompanied by bony outgrowths either in children or adults. Again, reference to the pathological appearances in Hale White's case of early rheumatoid arthritis will show a remarkable similarity between the morbid changes observed in it and Still's disease. In both instances the changes are largely those found in the synovial membrane and peri-articular tissues, while the cartilaginous erosions in both were slight in degree and referable to absorption by pressure of thickened synovial processes, but in neither case were there any osteophytic outgrowths. Again, in Whitman's case rarefaction of the shafts of the bones, as evidenced by the extremely thin cortex, was present, and this is exactly comparable to what has been observed in the shafts of the bone in rheumatoid arthritis of both children and adults.

Neither can any differentiation be established on the ground that splenic and lymphatic enlargement are absent in rheumatoid arthritis; indeed, the evidence of McCrae and other observers seems to indicate that enlargement of lymphatic glands, far from being absent, is very common in rheumatoid arthritis, while in addition McCrae noted splenic enlargement in four of his cases. To my mind, too much stress has been laid on glandular enlargement as a differentiating feature, for having regard to the tender age of the patients in Still's disease, there is nothing unusual that, in the presence of a toxic or infective condition, glandular or splenic enlargement should result, inasmuch as children in this respect react more markedly than adults. If no other point of differentiation can be found, then it is obvious that if we regard Still's disease as a form of infectious arthritis, as American authorities—Morse, Goldthwait, and others—do, we should, to be consistent, relegate also those forms of rheumatoid arthritis, whether occurring in children or adults, and attended by glandular enlargement, to the same category of infectious arthritis. The rarity of splenic enlargement in adult cases cannot be adduced as an argument against this view, for, as we have seen, it is by no means a constant feature in Still's disease. Hence it seems to me that we must look elsewhere for our basis of differentiation, and in default of bacteriological findings of a positive nature it is not easy to see how this can be achieved, for the muscular atrophy and other features are reproduced in the subjects of Still's disease.

In conclusion, therefore, it appears that pending further bacteriological research we are not in a position to state definitely, on the grounds above alluded to, that Still's disease is a distinct entity, and for the present I am inclined to regard it as a form of rheumatoid arthritis, in which the prominence of glandular enlargements is to be accounted for as a peculiarity incidental to the early age of the subjects, and that their presence is not necessarily an indication of its being a specific disease distinct from rheumatoid arthritis.

OSTEO-ARTHRITIS IN CHILDREN.

This condition, though extremely rare, has been met with in young children. Nothing is known as to its causation, if we except the observations made by Bowlby,[25] who noted that in a boy of nine years of age, the subject of hæmophilia, osteo-arthritic changes developed as the result of repeated intra-articular hæmorrhages. Apparently, generalized and localized forms of the disease can occur. Thus Eikenbury,[26] of Chicago, records a case in a boy aged eleven, in whom there was present widespread bony ankylosis of the joints implicating both elbows, both wrists, and several digits. The mobility of the shoulder and knee joints also was much restricted and attended by grating. In addition, both ankles and the temporo-maxillary and cervical articulations were ankylosed. The previous history revealed the fact that when seven years old he had injured his left hip. This was followed after some years by a severe multiple arthritis, attended by high fever and a history of hæmaturia. No suppuration followed, but he suffered with articular pains subsequently, and when the patient came under the observation of Eikenbury, he presented the above widespread articular disease. The osseous character of the lesions was demonstrated by skiagraphy.

In addition, Diamantberger also notes that generalized osteo-arthritis is to be met with in children. Of the localized forms, he cites four instances of the occurrence of Heberden's nodes in children. In view of the fact that the phalangeal nodosities, said by Bouchard to develop in cases with dilatation of the stomach, are with difficulty distinguished from the nodosities of Heberden, it is interesting to note that in three of the above examples the children suffered with gastro-intestinal disturbances attended by dilatation of the stomach. Still has recorded two cases of nodosities in children the subjects of congenital syphilis, the enlargements presenting very similar appearances to those described by Heberden. Preiser,[27] as before alluded to, records a case of so-called "idiopathic" juvenile arthritis deformans coxæ, and his monograph also includes a study of fourteen cases of the affection recorded by other German authors.

Gulliver[28] has also reported an example of this disease in the hip following trauma in a boy aged fifteen.

Pathologically, there is reason to believe, according to Hoffa[29] and others, that the osteo-arthritic changes in some instances are engrafted upon, and possibly related to, a previous inflammatory process of a tubercular nature in the affected joint.

I am unaware of any description of a post-mortem examination of this disease, so rare in childhood, with the exception of one recorded by Spitzy.

Hoffa, reviewing this case, maintains that the anatomical changes were undoubtedly those of osteo-arthritis.

The subject was a boy, aged six years and seven months, in whom a condition of chronic multiple arthritis had existed for three years. The cause of death was apparently lardaceous disease, complicated by long-standing dropsy.

There was no account given of changes in the synovial membrane.

On examination of the hip-joint, the upper surface of the head of the femur was segmented, uneven, and in places destitute of cartilage. The changes were more marked in the central portion, the underlying bony tissue having undergone rarefaction.

At the peripheral margin, on the contrary, there were present warty excrescences, consisting of hard, dense, bony tissue.

The acetabulum in one spot showed excavation, while around its brim were situated new bony formations.

Exactly similar changes were to be found in the knee and elbow joints.

CHRONIC FIBROUS RHEUMATISM.

The etiology and pathology of this condition, according to Still, are probably identical with those of acute rheumatism. The disease is very rare in children, and I have seen no example of it. Still records an instance, however, in a child three and a half years of age, in which there was no previous acute attack, and the disease arose insidiously. The joints, as before stated, show peri-articular thickening and no evidence of bony change. Deformities similar to those of rheumatoid arthritis may be evolved secondarily to invasion of related tendon sheaths by the fibrotic process. Enlargement of the glands or spleen is absent. There may, however, be rheumatic manifestations, such as valvular disease of the heart and subcutaneous fibroid nodules, as was noted in Still's case. I gather from Still's article that Brigidi and Banti[30] record an autopsy on a case in an adult in which the presence of fibrous change in the capsule and peri-articular tissues was ascertained, the cartilage, however, being unimplicated.

* * * * *

DIAGNOSIS.

The differential diagnosis of these four conditions as far as possible has been made apparent in the foregoing clinical description of them individually. It now only remains to allude to their possible confusion with other disorders.

As Diamantberger points out, in cases of rheumatoid arthritis with early spinal involvement, the associated muscular spasms may give rise to an erroneous diagnosis of spasmodic torticollis and tetany. He cites an instance in which the former diagnosis was made, and the infant succumbing shortly to an attack of enteric fever it was found that the cervical vertebræ were ankylosed and in part destroyed. In instances where the spine is involved early it is possible also to confound it with spinal caries. Grancher,[31] as far back as 1888, stated that it might be confused with such, and in addition Still issues a warning on this point.

The last-named author, when dealing with the diagnosis of the variety associated with his name, states that in the acute stage it is often mistaken for acute rheumatism, but is to be distinguished by the persistence of the joint swellings and splenic and glandular enlargement. When the knees are early affected the difficulty in walking, according to him, may lead to its confusion with rickets. Its confusion with tubercular disease of the type of ordinary white swelling is hardly likely, owing to the widespread character and symmetrical distribution of the articular lesions. In view of Edsall's work, however, it must be remembered that tubercular arthritis is not necessarily always of the type of *tumeur blanche*, and in cases of Still's disease with glandular enlargement the possibility of its being of this aberrant tubercular nature must be borne in mind. Again, similarly, the monarticular forms of osteo-arthritis must not too readily be ascribed to simple trauma, but the possibility of underlying tubercular mischief must be entertained. Finally, the joint swellings of congenital syphilis may present a close resemblance, but the history and the presence of other signs of syphilis will suffice to prevent error.

TREATMENT.

This can hardly be considered encouraging, having regard to their tender years and the inveterate nature of their affection.

The general measures advocated for the treatment of the disease in adults, such as abundance of nutritious food, as much sunshine as possible, and fresh air, are indicated. Similarly, the avoidance of cold damp sites is advisable.

The clothing should be warm but light, and as far as possible an outdoor life in a warm, dry climate should be pursued, and the child, in the absence of any acute mischief, should be encouraged to use his joints. The evil effects of contracture should be prevented as far as possible.

Arsenic, guaiacol carbonate, cod-liver oil, and syrup of iodide of iron are, as in the case of adults, applicable for the treatment of the affection. Still recommends hot-air baths, but in cases that have come under my care I have found thermal bathing efficient combined with massage both dry and wet. As we have seen, in the treatment of Still's disease, incision and washing out of the affected joints has been followed by recovery, as in Whitman's case before alluded to.

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